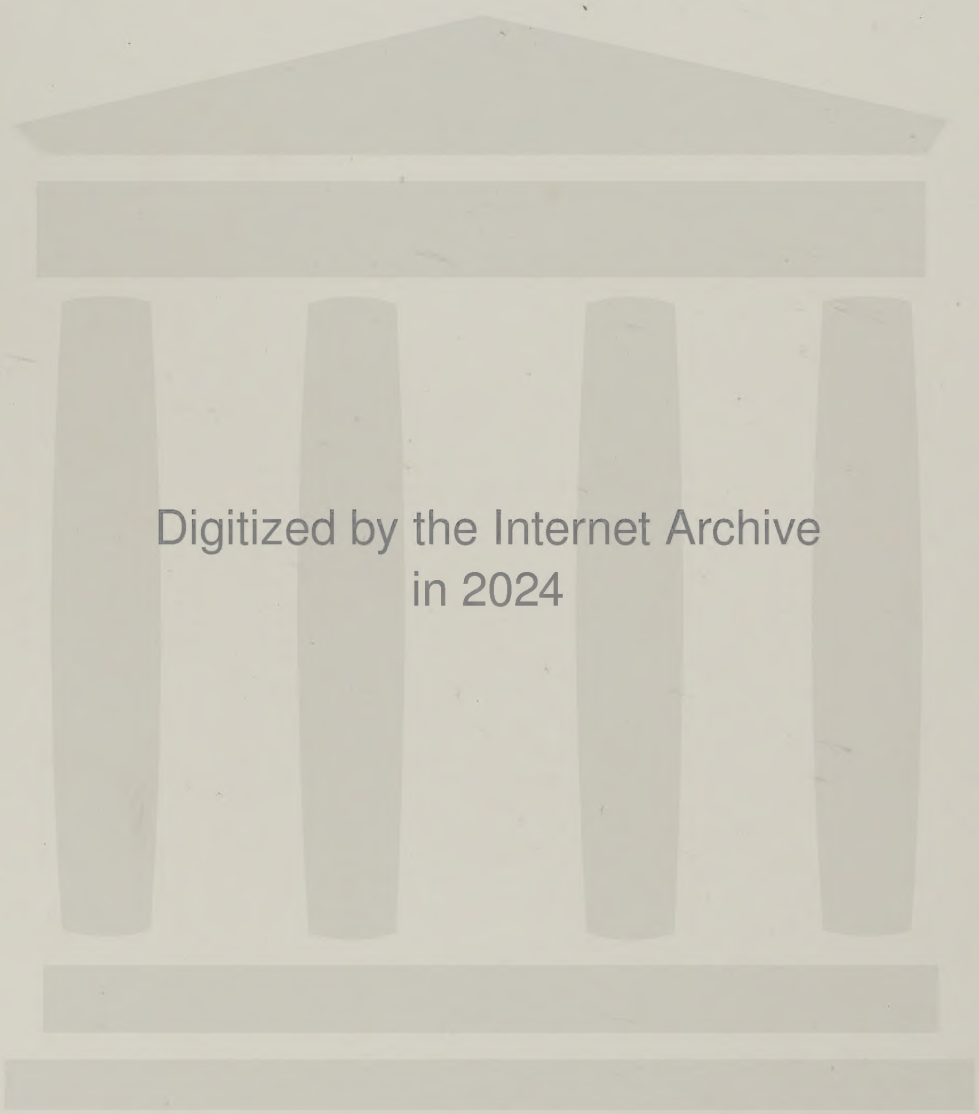
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SINUS THROMBOPHLEBITIS

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INFLAMMATORY DISEASES OF THE VENOUS SINUSES OF THE DURA MATER

BY

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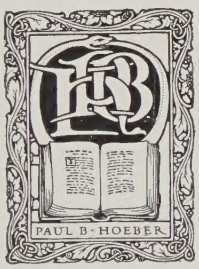

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TO MY DAUGHTER

SUSAN

PREFACE

GENERAL sepsis of otitic origin is one of the most important complications of middle-ear inflammation. It is of importance not only to the otologist, but to the internist and the pediatrist as well, for there are many borderline cases in which the ear symptoms are so slight that they are overlooked by the patient and the physician. The ear may appear normal or almost normal when the signs of sepsis set in, and the case may simulate typhoid, pneumonia or some other non-otologic condition. On the other hand, a general systemic disease may occur as an accidental complication of middle-ear suppuration, and may simulate otogenic general sepsis. Many of these patients are admitted to the hospital as general medical cases, and do not reach the otologist until very late in the disease, if at all. The recognition of otogenic general sepsis early in the disease is of very great importance, because proper surgical treatment in the early stages cures most of the patients. If left untreated, or if not treated until very late, most of the patients die.

The writer has endeavored to draw the clinical picture of otogenic general sepsis as clearly and as sharply as it is possible to delineate a condition which has such protean manifestations. He has attempted to indicate the usual and some of the unusual forms which the disease may assume. He has tried to correlate the clinical picture with the underlying pathological condition, and to formulate a rational treatment, based upon the anatomy and the pathological changes in the tissues involved.

The writer has included in this work only those opinions which have seemed to him to be firmly founded on fact, in order not to complicate the issues unnecessarily.

The last few years have brought a number of valuable adjuvants to the diagnosis and the treatment of otogenic general sepsis. If the presenta-

tion of the facts in this work will lead to further stimulation of thought along these lines, the writer will feel amply repaid for the effort expended.

The illustrations in this work were all drawn by the author. Most of them are original. Some of them were modified from other illustrations.

The writer wishes to thank Mr. Paul B. Hoeber and his able staff for the careful and painstaking manner in which they have prepared this volume.

ALFRED BRAUN

NEW YORK, N. Y.,
July, 1928.

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Chapter I

DEVELOPMENT

SINUS THROMBOPHLEBITIS

Chapter I

DEVELOPMENT

THE first indication of blood vessels in the head of a chick is seen thirty-eight to forty-two hours after incubation. A capillary sprout grows from the arch of the aorta, which at this age is situated near the root of the optic vesicle. This capillary sprout at first has no circulation, since it has no connection with the venous end of the heart.

A small branch is soon formed which leaves the lateral surface of the aorta opposite the venous end of the heart, and passes around to the vitelline veins on either side. This tiny vessel is the beginning of the duct of Cuvier. The capillaries in the head join the duct of Cuvier, and their connection with the aorta is broken. In this way a circulation is established from the head, through the duct of Cuvier, to the venous end of the heart.

At the same time, a small vessel from the trunk joins the duct of Cuvier at the point where the latter receives the vessel from the head. There is a duct of Cuvier on either side of the body, the two vessels joining and forming the sinus venosus of the heart. The ducts of Cuvier eventually become the innominate veins (Fig. 1). The branch of the duct of Cuvier from the trunk is called the posterior cardinal vein.

The capillary sprout which enters the head becomes a capillary network, which surrounds the primitive neural tube. This capillary network finally fuses into two veins, one of which lies on either side of the neural tube. They lie dorsal to the optic vesicle, and ventral to the otic vesicle. They start at the forebrain and pass backward medial to the cranial nerves, between the nerves and the neural tube. These vessels are called the anterior cardinal veins or primary head veins.

In a human embryo 3 mm. long, there is found, in addition to the vein which lies medial to the cranial nerves, another vein which is lateral to the nerves (Fig. 2). This second vein starts at the level of the acoustico-facial

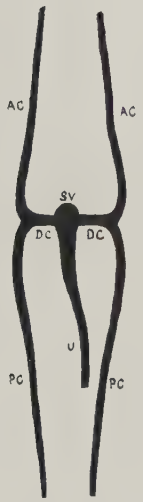

FIG. 1. CARDINAL VEINS. (*After Koelliker.*)

AC, anterior cardinal vein; DC, duct of Cuvier; PC, posterior cardinal vein; SV, sinus venosus; U, umbilical vein.

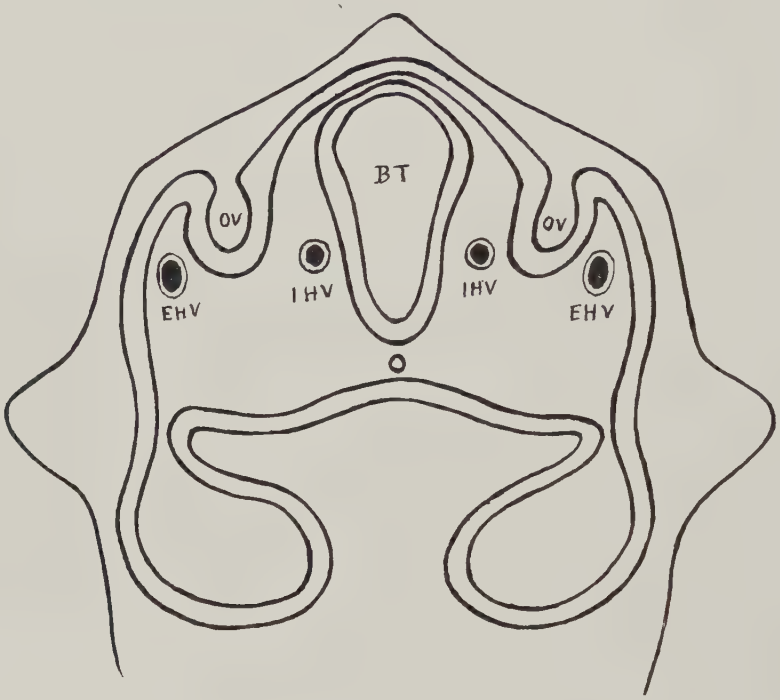

FIG. 2. TRANSVERSE SECTION THROUGH HUMAN EMBRYO 3 MM. LONG. (*After Sabin.*)

BT, brain tube; EHV, lateral portion of primary head vein; IHV, medial portion of primary head vein; OV, otic vesicle.

nerves, passes backward to the lateral side of the vagus, and finally joins the duct of Cuvier. Anastomoses form at various points along the course of these two veins, forming rings of veins around the cranial nerves. In places, the medial portions of the rings disappear, and the lateral portions remain. In this way, the portion of the primary head vein which extends from the fifth to the tenth cranial nerves comes to lie to the outer side of the nerves. The portion of the medial vessel which extends from its anterior extremity

to the region of the trigeminal nerve remains. From here to the eleventh and twelfth nerves, the medial vein disappears, and the primary head vein lies external to the cranial nerves and the otic vesicle, while opposite the eleventh

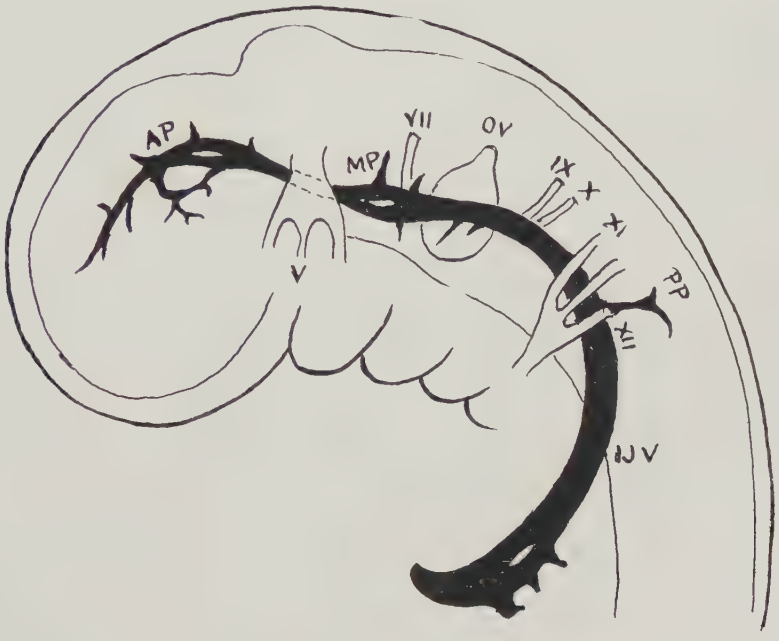

FIG. 3. LONGITUDINAL SECTION OF HUMAN EMBRYO 4 MM. LONG, SHOWING PRIMARY HEAD VEIN. (*Modified after Streeter.*)

AP, anterior dural plexus; IJV, portion of primary head vein which eventually becomes internal jugular vein; MP, middle dural plexus; OV, otic vesicle; PP, posterior dural plexus; V, trigeminal ganglion; VII, facial nerve; IX, glossopharyngeal nerve; X, pneumogastric nerve; XI, spinal accessory nerve; XII, hypoglossal nerve.

and twelfth nerves, it again lies medial to the nerves. It then passes lateral to the ascending aorta, and empties into the duct of Cuvier. In other words, the beginning and the end of the primary head vein remains medial to the cranial nerves, while the middle portion comes to lie lateral to the nerves (Fig. 3).

The primary head vein is a vessel consisting of a single layer of endothelial cells. It receives tributaries from the forebrain, interbrain and midbrain. These branches lie in the depressions of the brain vesicle.

The dorsal tributaries to the primary head vein are divided into three groups, anterior, middle and posterior. The anterior group empties into the primary head vein in the region of the anterior and middle cerebral vesicles.

FIG. 4. HUMAN EMBRYO 14 MM. LONG. (*Modified after Streeter.*)

AP, anterior dural plexus; IJV, internal jugular vein; IX, glossopharyngeal nerve; MP, middle dural plexus; OV, otic vesicle; PP, posterior dural plexus; V, trigeminal ganglion; VII, facial nerve; X, pneumogastric nerve; XI, spinal accessory nerve; XII, hypoglossal nerve.

The middle group empties into the primary head vein in the region between the trigeminal nerve and the acoustico-facial complex. The posterior group empties into the primary head vein in the neighborhood of the vagus rootlets.

These three tributaries form rich plexuses of veins, and are called the *anterior dural plexus, the middle dural plexus* and the *posterior dural plexus* (Fig. 4).

From the ventral side of the main venous trunk, deeper lying plexuses are given off, from which are developed the cerebral veins, which drain the brain tissue.

Divisions of Primary Head Vein. The main trunk of the primary head vein may be divided into three portions: (1) the anterior segment, which is the portion anterior to the semilunar ganglion; (2) the middle segment which is the portion between the semilunar ganglion and the mouth of the posterior dural plexus; and (3) the posterior segment which is the portion between the posterior dural plexus and the duct of Cuvier. The anterior segment develops into the cavernous sinus, and retains its position medial to the fifth nerve throughout the entire period of development. It collects blood from the forebrain, interbrain and midbrain. The middle segment disappears after a while, and is replaced by a more dorsally situated channel, which becomes the lateral sinus, and collects blood from the hindbrain and afterbrain. The posterior segment bends downward and becomes the internal jugular vein.

The *anterior segment* receives four tributaries, the anterior dural plexus, the ophthalmic vein, the anterior cerebral veins and the sphenoparietal sinus. The ophthalmic vein lies medial to the maxillary division of the trigeminal nerve, and it drains the region of the optic vesicle.

As the human embryo becomes a little older, for example, in an embryo about 18 mm. long, an anastomosing channel is formed dorsal to the otic capsule, and lateral to the endolymphatic sac (Fig. 5). This vessel unites the trunks of the middle and posterior dural plexuses, and this new channel (ls, Fig. 5) eventually becomes the horizontal portion of the lateral sinus. Most of the blood from the middle dural plexus now drains back into the

posterior dural plexus through this vessel. The trunk that originally connected the middle dural plexus to the primary head vein (M, Fig. 5) gradually diminishes in size, and finally degenerates into a minute plexus, which

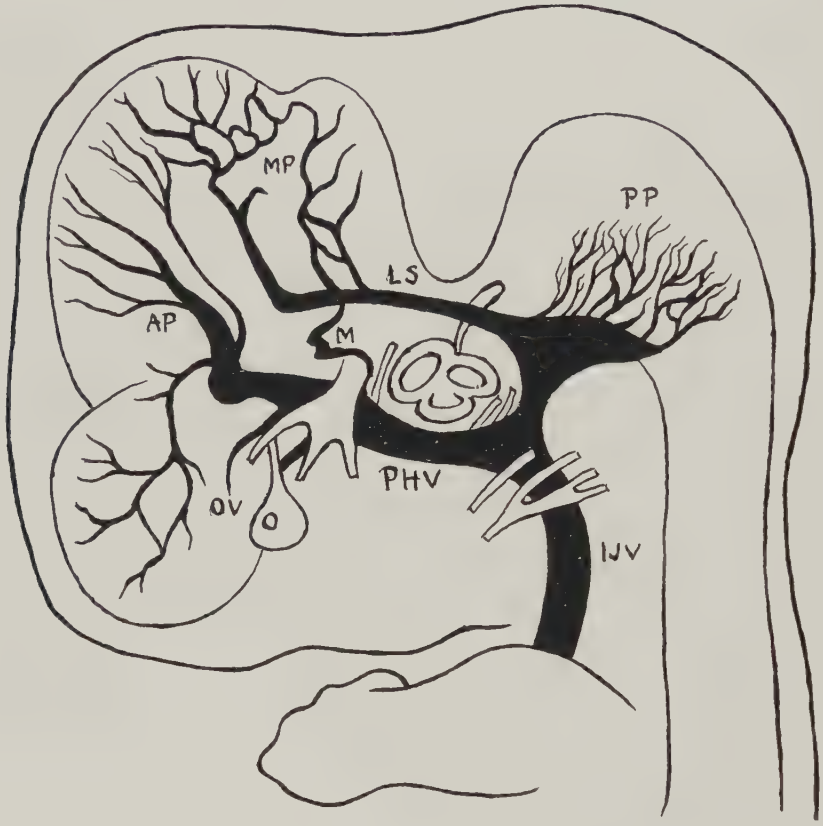

FIG. 5. HUMAN EMBRYO 18 MM. LONG. (*Modified after Streeter.*)

AP, anterior dural plexus; IJV, internal jugular vein; LS, lateral sinus; M, original trunk of middle dural plexus, which later becomes the superior petrosal sinus; MP, middle dural plexus; O, optic vesicle; OV, ophthalmic vein; PHV, primary head vein; PP, posterior dural plexus.

carries off a very small part of the blood from the middle dural plexus. This small plexus of veins, which was originally the trunk of the middle dural plexus, again becomes a large vessel, at a later stage of development, and forms the future superior petrosal sinus. It passes from the anterior end of the lateral sinus to the posterior end of the cavernous sinus.

The original middle segment of the primary head vein, i.e., the portion between the trunks of the middle and posterior dural plexuses, now disappears, and the blood from the anterior and middle dural plexuses reaches

FIG. 6. HUMAN EMBRYO 21 MM. LONG. (*Modified after Streeter.*)

AP, anterior dural plexus; CS, cavernous sinus; ICV, inferior cerebral vein; IJV, internal jugular vein; LS, lateral sinus; OV, ophthalmic vein; PHV, remnant of otic portion of primary head vein, which originally connected cavernous sinus with internal jugular vein; PP, posterior dural plexus; SLS, superior longitudinal sinus; SPS, superior petrosal sinus; SS, sigmoid sinus; V, trigeminal ganglion.

the posterior segment of the primary head vein through the newly formed vessel which lies dorsal to the otic capsule, and through the trunk of the posterior dural plexus (Fig. 6).

The original *middle segment* of the primary head vein was situated, in part, external to the plane of the future bony skull, leaving the skull between the fifth and seventh nerves. This vessel sometimes persists after birth, and is then known as the petrosquamosal sinus. When it persists, it leaves the skull through a foramen which was first described by Luschka, and called by him the foramen jugulare spurium. This foramen is usually located in the glenoid fossa, near the Glaserian fissure, but it may be situated in the root of the zygoma. It was believed by Luschka that the blood which passed through this persistent petrosquamosal sinus entered the external jugular vein. He believed that in the early embryonic stage, before the lateral sinus was formed, the blood from the intracranial veins left the skull through the foramen jugulare spurium and emptied into the external jugular vein, and at a later stage, when the petrosquamosal sinus became obliterated, and the lateral sinus was formed, the blood was shunted into the internal jugular vein. This theory was accepted by embryologists for many years, until Salzer, in 1895, showed conclusively that the external jugular vein did not appear until long after the lateral sinus was formed. He proved beyond doubt that, in all stages of embryonic development, the internal jugular vein was the main channel of exit of the blood from the skull.

With the disappearance of the middle segment of the primary head vein, the cavernous sinus drains upward over the semilunar ganglion into the future lateral sinus, through the small channel which was originally the trunk of the middle dural plexus, and which finally develops into the superior petrosal sinus (SPS, Fig. 6).

The anterior and middle dural plexuses gradually come into closer anastomosis with each other. Only a small portion of the anterior plexus now

drains into the cavernous sinus. Most of the anterior plexus, together with the middle plexus, drains into the new lateral sinus which lies dorsal to the otic capsule.

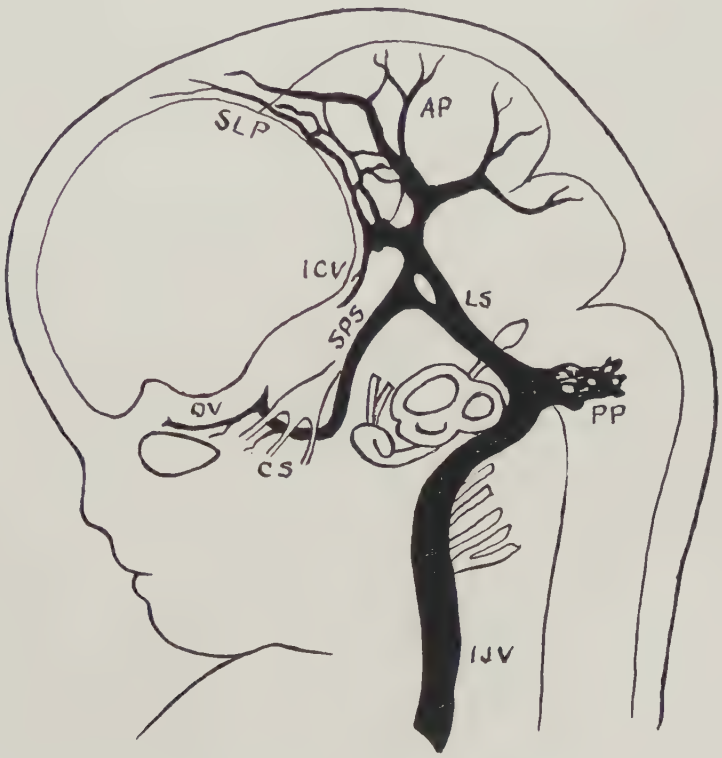

FIG. 7. HUMAN EMBRYO 24 MM. LONG. (*Modified after Streeter.*)

AP, anterior dural plexus; CS, cavernous sinus; ICV, inferior cerebral vein; IJV, internal jugular vein; LS, lateral sinus; SLP, superior longitudinal plexus; SPS, superior petrosal sinus; OV, ophthalmic vein; PP, posterior dural plexus.

The *posterior dural plexus* eventually becomes the occipital sinus, and drains the tentorium cerebelli, and part of the cerebellum (Fig. 7).

As the origin of the bony skull is indicated, it is seen that the primary head vein perforates the skull through an opening which is to be the future jugular foramen (Fig. 8). The portion of the primary head vein immediately above this foramen is the sigmoid portion of the lateral sinus, and the part

between the jugular foramen and the duct of Cuvier is the internal jugular vein. The sigmoid sinus curves around the otic capsule and occupies about the same position as in the adult skull.

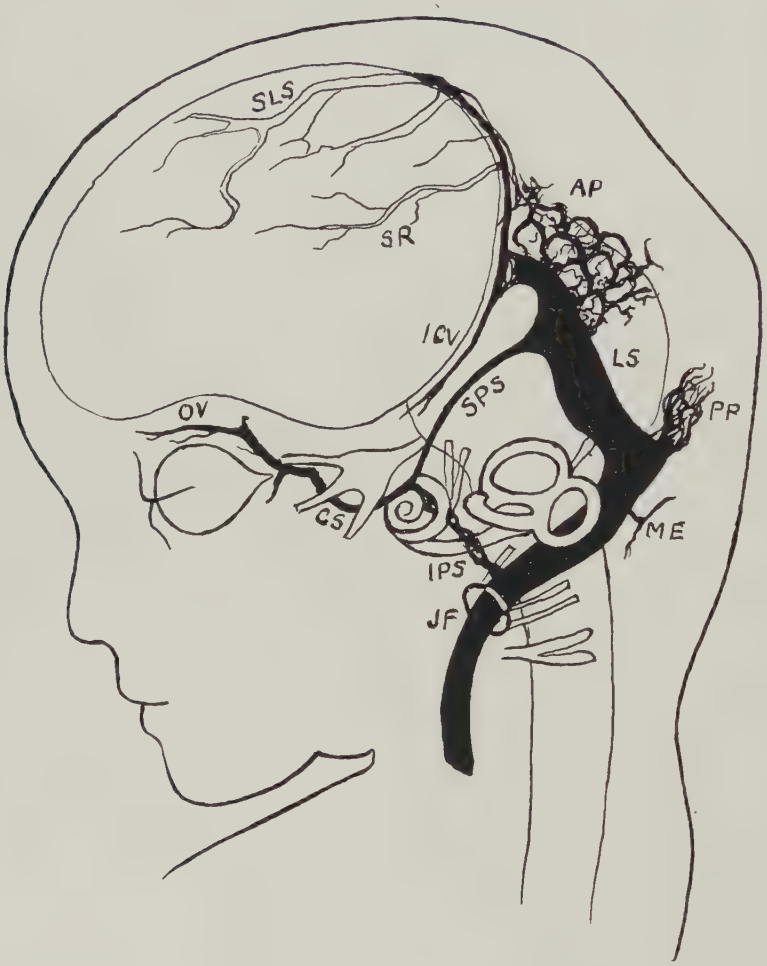

FIG. 8. HUMAN EMBRYO 50 MM. LONG. (*Modified after Streeter.*)

AP, anterior dural plexus; CS, cavernous sinus; ICV, inferior cerebral vein; IPS, inferior petrosal sinus; JF, jugular foramen; LS, lateral sinus; ME, mastoid emissary; OV, ophthalmic vein; PP, posterior dural plexus; SLS, superior longitudinal sinus; SPS, superior petrosal sinus; SR, sinus rectus.

The plexus of vessels which constitutes the remnant of the trunk of the middle dural plexus gradually develops into the superior petrosal sinus. It

passes over the cochlear part of the otic capsule, and empties above into the lateral sinus (SPS, Figs. 7 and 8).

Another plexus of vessels is now formed, which extends from the

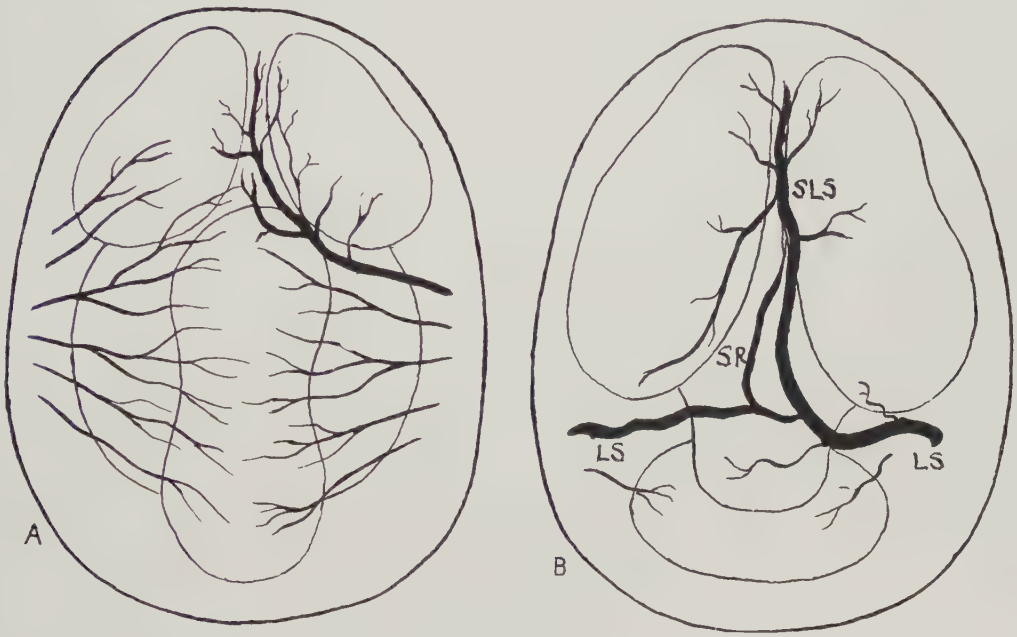

FIG. 9. A. VERTEX VIEW OF HUMAN EMBRYO 20 MM. LONG. B. VERTEX VIEW OF HUMAN EMBRYO 54 MM. LONG. THESE FIGURES SHOW THE CONVERSION OF THE SUPERIOR LONGITUDINAL PLEXUS INTO THE SUPERIOR LONGITUDINAL AND STRAIGHT SINUSES. (*Modified after Streeter.*)

LS, lateral sinus; SLS, superior longitudinal sinus; SR, sinus rectus.

posterior end of the cavernous sinus, median to the otic capsule, and empties into the internal jugular vein just as the latter emerges from the jugular foramen. This vascular plexus becomes the inferior petrosal sinus (IPS, Fig. 8).

The portion of the lateral sinus between the point of entrance of the superior petrosal sinus and the jugular foramen, i.e., the sigmoid portion, is the first part to be developed as a single large channel. The part of the lateral sinus anterior to the superior petrosal remains in the formative stage for some time longer, as is shown by the large capillary meshwork along its

dorsal margin. As the cerebral hemispheres grow larger, this channel migrates backward, and comes to assume a more and more horizontal course.

A fold of dura is now interposed between the two hemispheres in the region of the fore brain, which develops into the falx cerebri. It is directly continuous with the tentorium cerebelli. Some of the branches of the anterior and middle dural plexuses grow into this fold of dura, and eventually form the superior and inferior longitudinal and the straight sinuses (Fig. 8).

At first these dural plexuses are seen as two irregular meshworks' one on either side of the midline. The branches gradually approach the middle line. They merge at first anteriorly, and then posteriorly, some of the branches dipping down into the dural fold of the falx cerebri. Two vessels are gradually formed by a fusion of this dural meshwork, one lying above the other; the upper forms the superior longitudinal sinus and the lower, the straight sinus (Fig. 9). The latter has two converging limbs in front that drain the choroidal masses of the cerebral hemispheres.

Variations at the Torcular. The superior longitudinal sinus and the straight sinus gradually grow backward, as the cerebral hemispheres enlarge. This development is accomplished at the expense of the anterior and middle dural plexuses. The channel repeatedly shifts into a more caudal loop of the plexus, the new loop enlarging and the old loop gradually diminishing in size and finally disappearing. The superior longitudinal and the straight sinuses empty into the lateral sinuses behind. The confluens sinuum or torcular Herophili is the point at which this caudal development reaches its completion.

Differences in Individuals. The differences in the arrangement of the sinuses at the torcular in different individuals is the result of variations in the shifting of the loops of the dural plexuses in the development of the superior longitudinal and straight sinuses. This region usually retains a trace of the plexiform character that is found in the embryonic stage. The asym-

metrical termination of these sinuses is seen, in the embryo as well as in the adult, by a tendency to drain more to one side of the head than to the other. In the embryos examined by Streeter, the superior longitudinal sinus drained

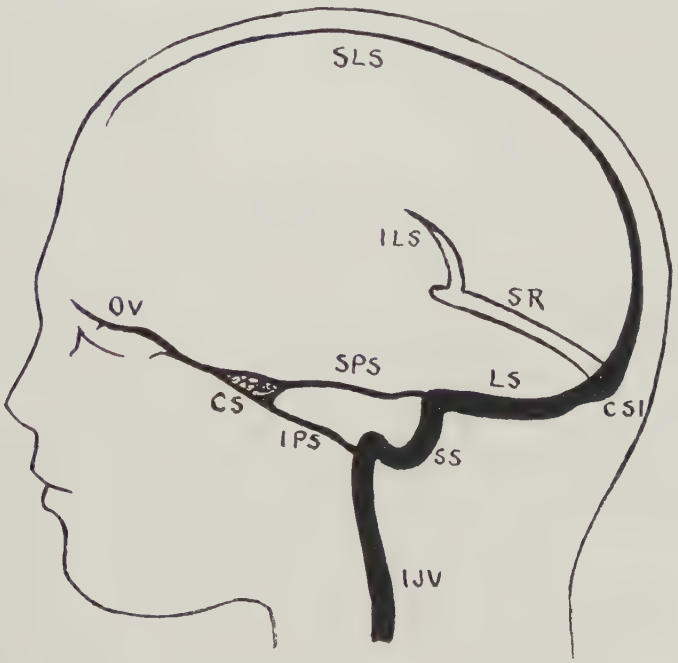

FIG. 10. ARRANGEMENT OF SINUSES IN ADULT. (*Modified after Streeter.*)

CS, cavernous sinus; CSI, confluens sinuum; IJV, internal jugular vein; ILS, inferior longitudinal sinus; IPS, inferior petrosal sinus; LS, lateral sinus; OV, ophthalmic vein; SLS, superior longitudinal sinus; SPS, superior petrosal sinus; SR, straight sinus; SS, sigmoid sinus.

predominantly toward the right lateral sinus in 89 per cent of the cases.

Diploic Veins. As the bony skull develops, branches are given off from the dural system of veins, which drain the bones of the skull. These become the diploic veins.

Vessels Draining Brain Tissue. In addition to the dural system of veins, two other venous systems are developed in the head; a system of deep vessels which drains the brain tissue, and a superficial system which drains the skin and superficial tissues. These three systems are all outgrowths of the same capillary plexus.

In the adult, the *superficial veins* maintain a few anastomoses with the dural system. These anastomoses constitute the emissary veins.

The cerebral veins drain into the dural sinuses, and their blood leaves the skull by means of the internal jugular vein.

The superficial system of veins begins with a vessel which drains the blood from the face. This vessel starts at the inner orbital margin and joins the internal jugular vein after the latter leaves the skull. This is the anterior facial vein. Another vessel drains the outer ear and the scalp, and joins the anterior facial at the angle of the jaw. The second vessel is the posterior facial vein. Another vein receives the blood from the deep portion of the facial skeleton, and joins the anterior facial vein after the latter passes the lower jaw.

There is a communication between the facial vein and the intracranial veins at the inner angle of the orbit. The vein which lies to the medial side of the lower jaw forms an anastomosis with the orbital veins. The external jugular vein gradually takes up part of the work of the internal jugular vein, and eventually the former vein forms the chief drainage for the superficial venous system of the head.

Chapter II

ANATOMY

Chapter II

ANATOMY

Venous Circulation of the Head

THE venous circulation of the head may be divided into four systems. The first or most superficial of these systems drains the skin and the soft tissues outside of the bony skull. The second system drains the bones of the skull. The third system drains the membranes of the brain, and the last and deepest system drains the brain tissue. The first system leaves the head chiefly by way of the external jugular veins, and to a lesser extent through the internal jugular veins. The remaining three systems leave the head principally by way of the internal jugular veins, and to a lesser degree, through various anastomoses, via the vertebral and external jugular veins (Fig. 11).

1. *Facial, temporal, internal maxillary, posterior auricular and occipital veins and their tributaries.* The first or most superficial system of veins consists of the facial, temporal, internal maxillary, posterior auricular and occipital veins, and their tributaries.

2. *Diploic Veins.* The venous system which drains the bones of the skull consists of the diploic veins.

3. *Meningeal Veins; Venous Sinuses of Dura.* The system which drains the membranes of the brain, consists of the meningeal veins and the venous sinuses of the dura.

4. *Cerebral Veins.* The system which drains the brain tissue is composed of the cerebral veins.

The second and fourth systems, i.e., the diploic and cerebral veins, pass into the venous sinuses before leaving the skull.

The sinuses communicate with the superficial veins of the head through vessels which perforate the skull. These vessels are called emissary veins.

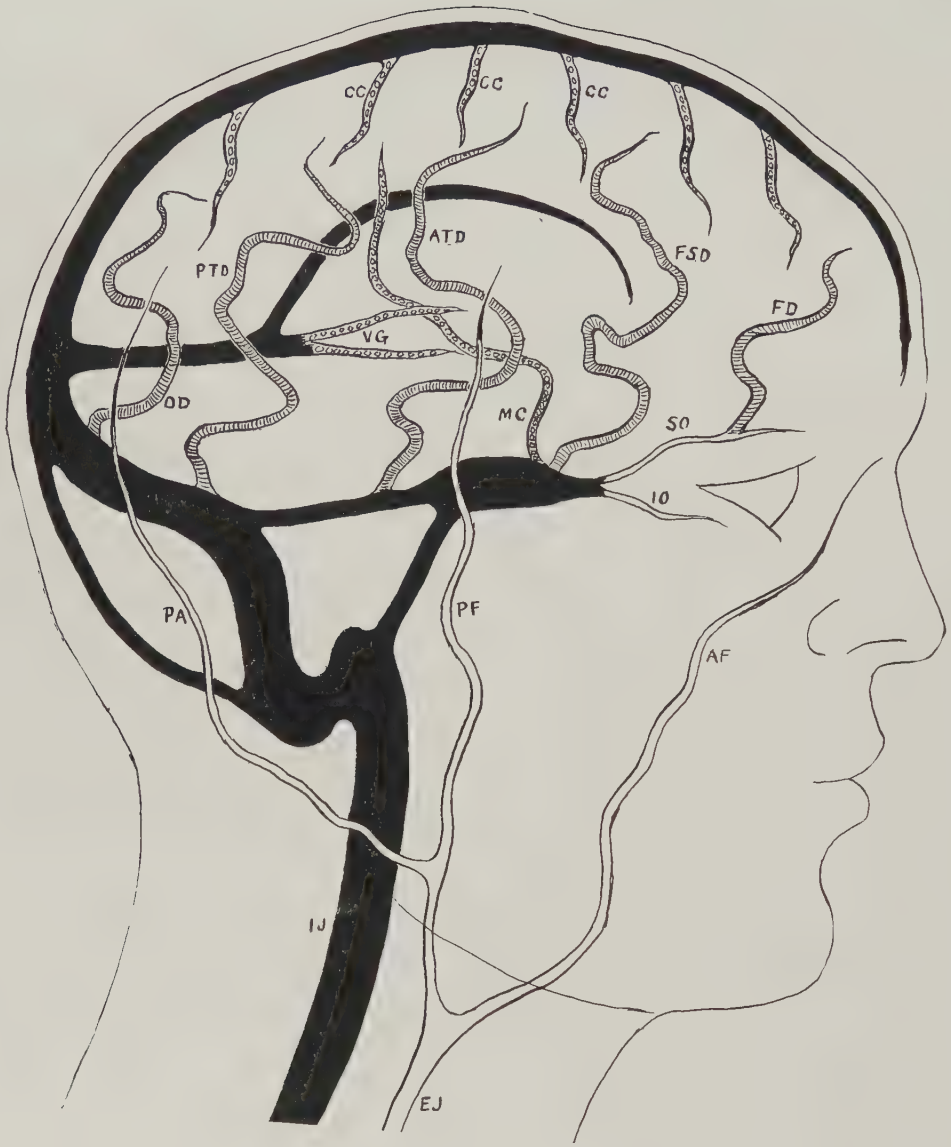

FIG. 11. DIAGRAM SHOWING FOUR VENOUS SYSTEMS OF HEAD. FROM WITHOUT INWARD, THEY ARE: 1, VEINS OF SUPERFICIAL TISSUES; 2, DIPLOIC VEINS; 3, VENOUS SINUSES OF DURA; 4, CEREBRAL VEINS.

Superficial veins ——; Diploic veins ||||||; Venous sinuses ■■■; Cerebral veins oooo.

AF, anterior facial vein; ATD, anterior temporal diploic vein; CC, superior cerebral vein; EJ, external jugular vein; FD, frontal diploic vein; FSD, frontosphenoidal diploic vein; IJ, internal jugular vein; IO, inferior orbital vein; MC, middle cerebral vein; OD, occipital diploic vein; PA, posterior auricular vein; PF, posterior facial vein; PTD, posterior temporal diploic vein; SO, superior orbital vein; VG, veins of Galen.

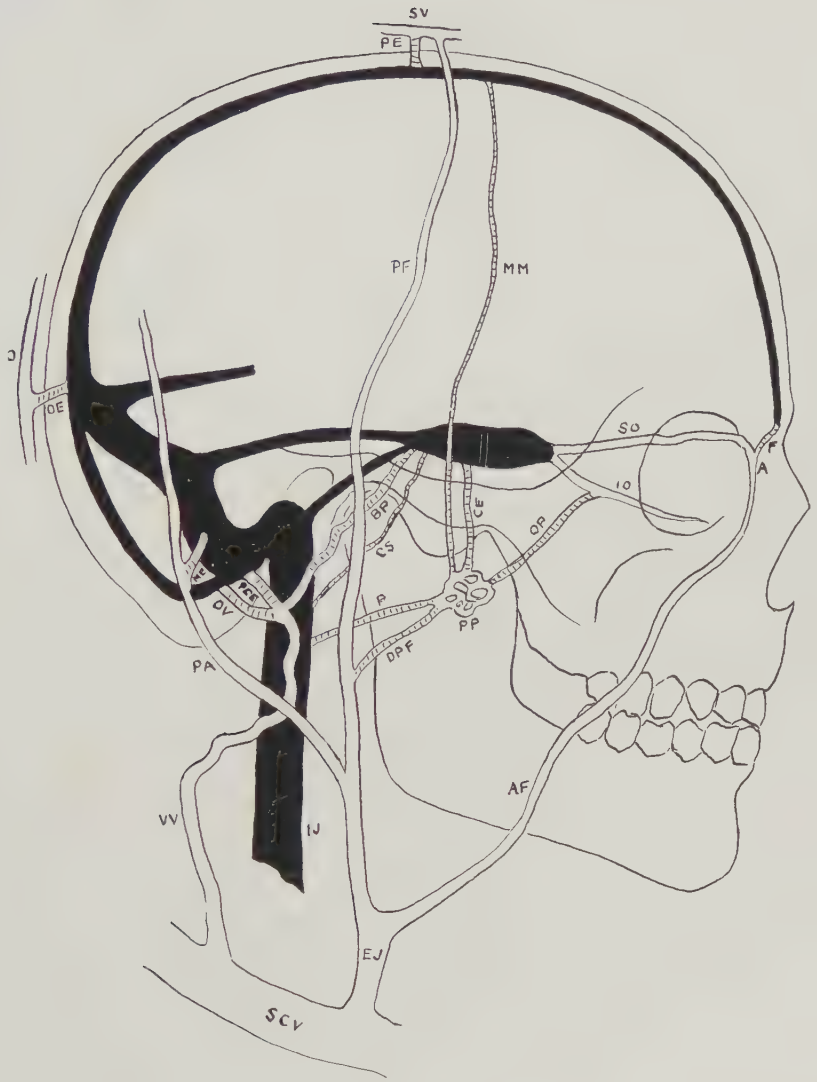

FIG. 12. DIAGRAM SHOWING COMMUNICATIONS BETWEEN SINUSES AND SUPERFICIAL VEINS.

Sinuses ▬; Superficial veins ——; Communicating veins ▥.

A, angular vein; AF, anterior facial; BP, basilar plexus; CE, communication between cavernous sinus and pterygoid plexus; CS, carotid sinus; DPF, deep posterior facial vein; EJ, external jugular; F, frontal vein; IJ, internal jugular; IO, infra-orbital vein; ME, mastoid emissary; MM, middle meningeal vein; O, occipital vein; OE, occipital emissary; OP, communication between infraorbital vein and pterygoid plexus; OV, communication between occipital sinus and vertebral vein; P, pharyngeal vein; PA, posterior auricular; PCE, posterior condyloid emissary; PE, parietal emissary; PF, posterior facial; PP, pterygoid plexus; SCV, subclavian vein; SO, supraorbital vein; SV, scalp vein; VV, vertebral vein.

There are a number of these vessels, the principal ones being the parietal emissary veins of Santorini, the mastoid emissaries, the occipital emissaries, and the condyloid emissaries. In addition to the emissary veins, the sinuses

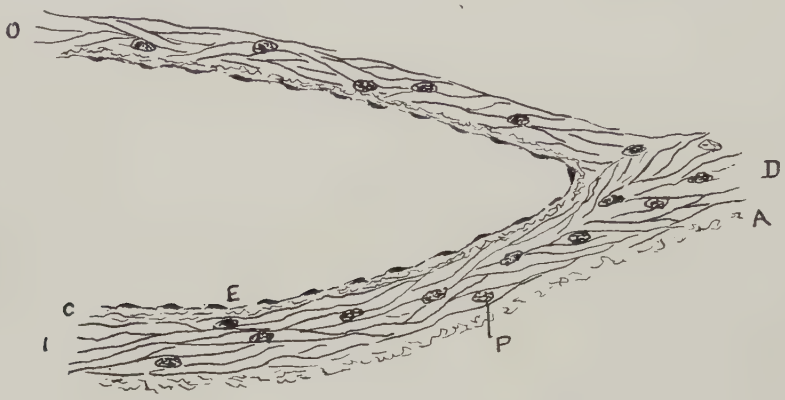

FIG. 13. CROSS-SECTION OF SINUS WALL.

A, arachnoid; C, delicate connective tissue lying under endothelial cells; D, dura; E, endothelial lining; I, inner sinus wall; O, outer sinus wall; P, plasma cells in connective tissue.

communicate with the superficial circulation through the vertebral veins, the ophthalmic veins, the middle meningeal veins, the pterygoid plexus, the basilar plexus and the carotid plexus (Fig. 12,)

The venous sinuses of the skull are intradural. The dura is split into two layers to enclose the sinuses. The outer is the thinner of the two layers, the proportion being about 1 to 2. For this reason, the outer sinus wall offers less resistance to an overlying infection than any other part of the dura. Some of the sinuses are in contact with the inner surface of the skull, as, for example, the lateral and superior longitudinal sinuses. Other sinuses lie some distance from the skull. The inferior longitudinal and straight sinuses are examples of the latter class.

The sinuses are rigid tubes, which cannot collapse, except when a portion of the bony skull is removed, exposing the sinus wall. There are no muscle fibers in the sinus walls. The sinuses possess no valves. The sinus walls are composed of the fibrous tissue which constitutes the dura, and

they are lined by a layer of endothelial cells, resting on an elastic bed of fine connective tissue (Fig. 13).

Here and there, along the course of some of the sinuses, especially

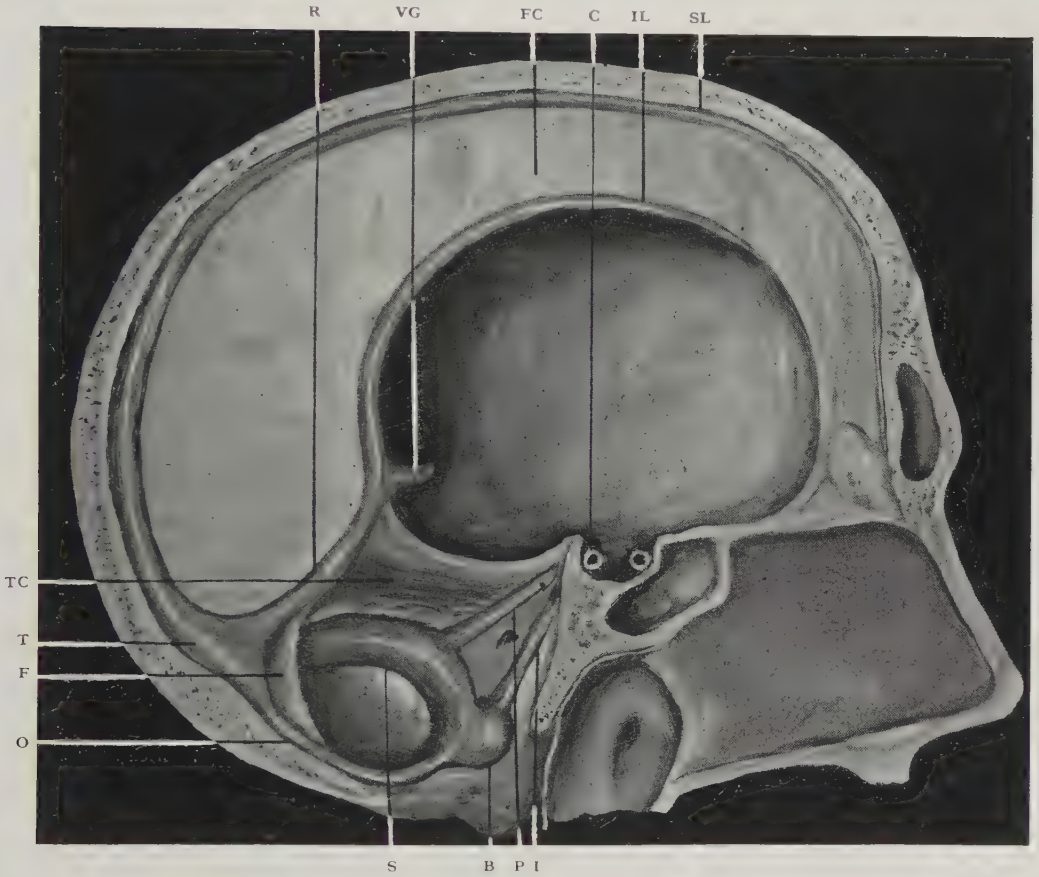

FIG. 14. SAGITTAL SECTION OF SKULL SHOWING VENOUS SINUSES.

B, jugular bulb; C, circular sinus; FC, falx cerebri; I, inferior petrosal sinus; IL, inferior longitudinal sinus; O, occipital sinus; P, superior petrosal sinus; R, straight sinus; S, sigmoid sinus; SL, superior longitudinal sinus; T, torcular; falx cerebelli; TC, tentorium cerebelli; VG, vein of Galen.

along the superior longitudinal, the lateral and straight sinuses, there are lacunar cavities, hollowed out of the thickness of the dura. They are called blood-lakes. They receive meningeal and diploic veins, and communicate with the cerebral veins.

The sinuses may be divided into two sets, those at the vault of the skull, and those at the base. In the sinuses at the vault are included:

1. The superior longitudinal sinus

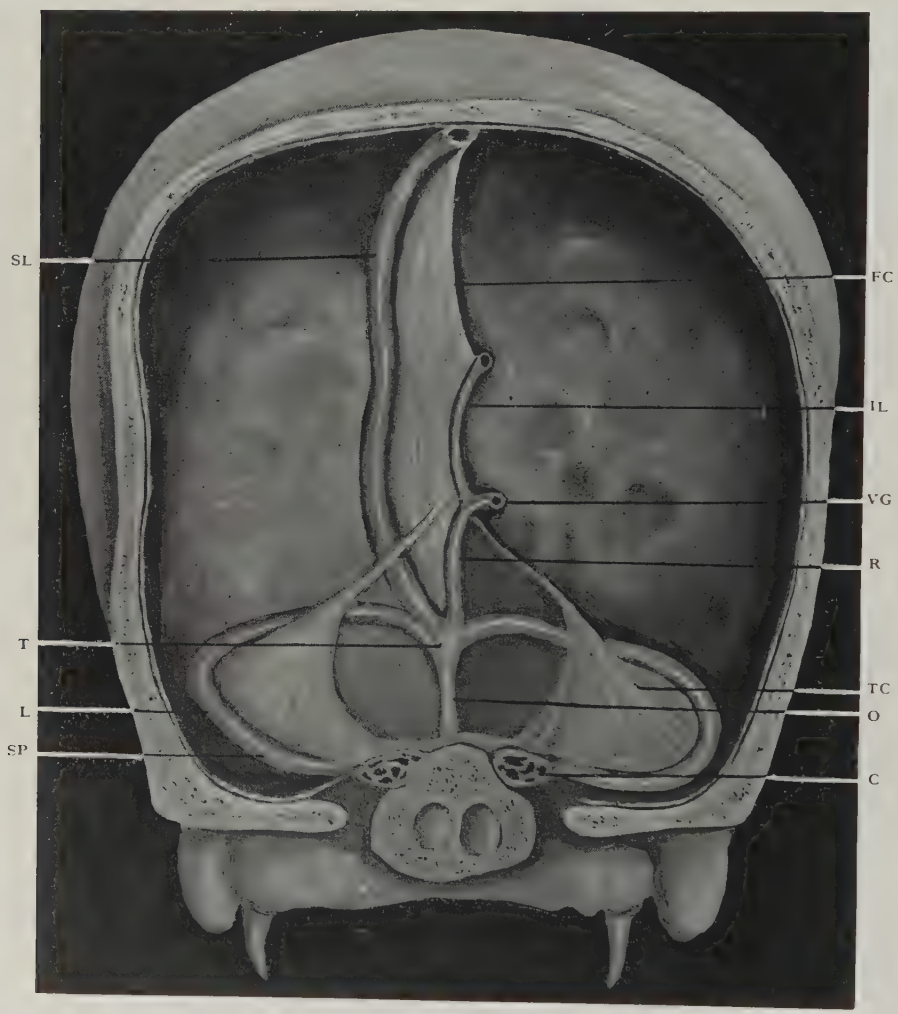

FIG. 15. CORONAL SECTION OF SKULL, AT LEVEL OF FORAMEN OVALE, LOOKING FROM BEFORE BACKWARD. (*After Henle.*)

C, cavernous sinus; FC, falx cerebri; IL, inferior longitudinal sinus; L, lateral sinus; O, occipital sinus; R, straight sinus; SL, superior longitudinal sinus; SP, superior petrosal sinus; T, torcular; TC, tentorium cerebelli; VG, vena Galeni.

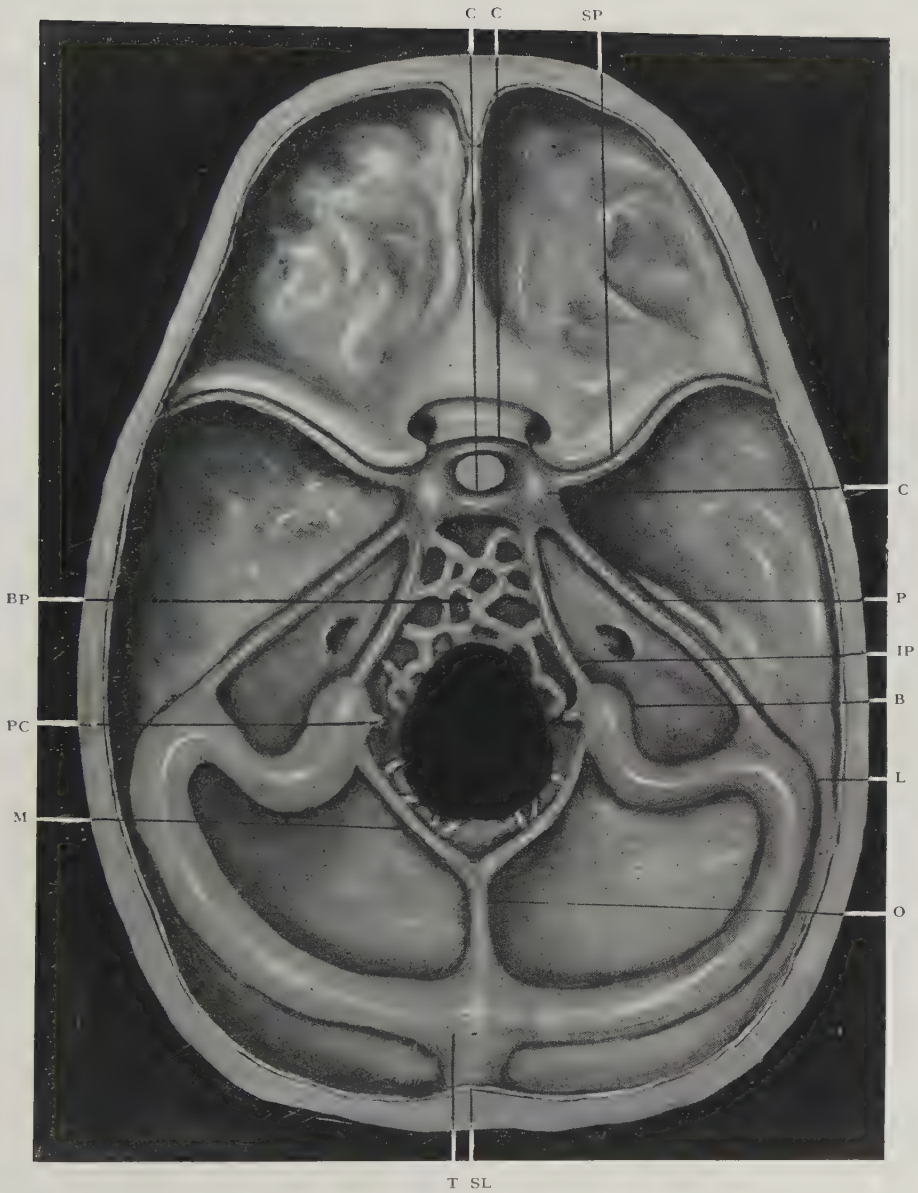

FIG. 16. HORIZONTAL SECTION OF SKULL, SHOWING VENOUS SINUSES AT BASE.

B, jugular bulb; BP, basilar plexus; C, cavernous sinus and circular sinus; IP, inferior petrosal sinus; L, lateral sinus; M, marginal sinus; O, occipital sinus; P, superior petrosal sinus; PC, posterior condyloid vein; SL, superior longitudinal sinus; SP, sphenoparietal sinus; T, torcular.

2. The inferior longitudinal sinus
3. The straight sinus (Figs. 14 and 15).

The sinuses at the base consist of the following:

1. The lateral sinuses
2. The posterior occipital sinuses
3. The superior petrosal sinuses
4. The inferior petrosal sinuses
5. The cavernous sinuses
   a. The circular sinus
   b. The sphenoparietal sinuses
6. The carotid sinuses
7. The anterior occipital sinus (Fig. 16).

In addition to these, there are several sinuses which occur only occasionally, such as the petrosquamous and the ophthalmopetrous sinuses.

Some of these sinuses are single, and some are paired. The single sinuses are the superior longitudinal, the inferior longitudinal, the straight, the posterior occipital (usually), the anterior occipital and the circular sinuses. The paired sinuses are the lateral, the superior petrosal, the inferior petrosal, the cavernous, the sphenoparietal and the carotid sinuses.

Sinuses at Vault of Skull

The Superior Longitudinal Sinus. The superior longitudinal sinus is a sickle-shaped vessel, which extends anteroposteriorly in the midline of the vault of the skull. It is imbedded in the upper or attached margin of the falx cerebri. It extends from the crista Galli, in front, to the torcular Herophili, behind. It is in contact with the frontal bone, the interparietal suture, and the occipital bone, terminating at the internal occipial protuberance. The sinus is triangular on cross section, the base of the triangle being

above. It is quite narrow, in front, and gradually increases in caliber toward its posterior end. Its diameter is 1 to 2 mm. in front, and 8 to 9 mm. at its posterior extremity. It is rather firmly attached to the overlying bone, and is easily torn when an attempt is made to expose it by removing its bony covering.

The Pacchionian bodies are vascular structures which take their origin from the arachnoid, and lie against the inner surface of the dura. In some places, they pierce the wall of the superior longitudinal sinus, and enter the lumen of the latter. They probably help in the regulation of intracranial pressure.

The superior longitudinal sinus receives blood from the inner and outer surfaces of the cerebral hemispheres. The veins from the frontal lobes of the brain enter the sinus obliquely from before backward, that is, in the direction of the blood current in the superior longitudinal sinus. Some of these branches enter at right angles to the sinus. The veins from the parietal and occipital lobes of the brain enter the sinus obliquely from behind forward, so that their blood stream is directed against the current in the sinus. This mechanism serves to slow the blood current in the superior longitudinal sinus (Fig. 17).

The diploic veins of the frontal and parietal bones also empty into the superior longitudinal sinus. These diploic veins enlarge into diverticuli before they enter the sinus. The middle meningeal veins communicate with these diverticuli, and act as an anastomotic chain between the superior longitudinal sinus and the pterygoid plexus. The pterygoid plexus empties into the external and internal jugular veins.

The middle cerebral vein and the great anastomotic vein of Trolard form a chain which connects the superior longitudinal sinus with the sinuses at the base of the skull.

The superior longitudinal sinus communicates with the veins of the nasal septum through a small vein which passes through the foramen

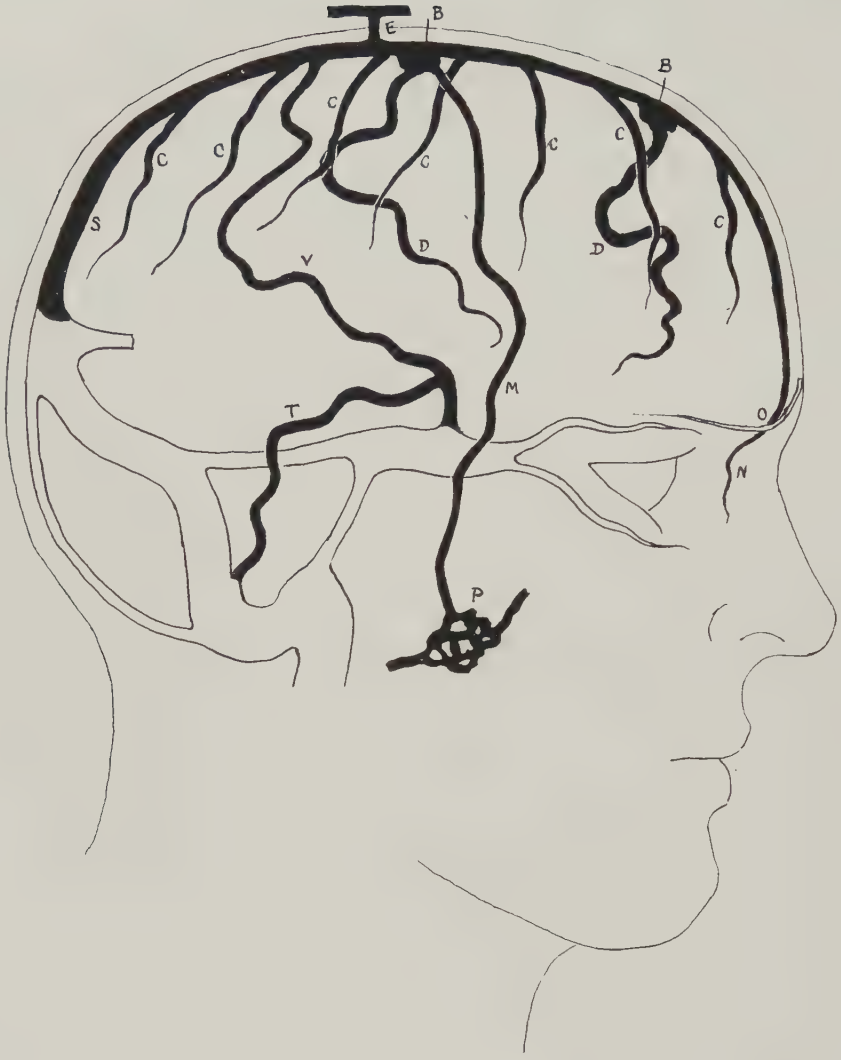

FIG. 17. TRIBUTARIES AND COMMUNICATIONS OF SUPERIOR LONGITUDINAL SINUS.

B, blood lakes; C, veins from frontal, parietal and occipital lobes; D, diploic veins; E, parietal emissary vein; M, middle meningeal vein; N, septal vein; O, communication between superior longitudinal sinus and septal vein through foramen cecum; P, pterygoid plexus; S, superior longitudinal sinus; T, great anastomotic vein of Trolard; V, middle cerebral vein.

cecum. This channel may transmit infections from the nose and nasal accessory sinuses to the superior longitudinal sinus.

The superior longitudinal sinus communicates with the veins of the scalp through parietal emissary veins which pass through the foramina of Santorini. The principal one of these emissaries is situated at the vertex of the skull near the posterior part of the interparietal suture. Through these veins, an infection of the scalp may extend into the sinus.

The superior longitudinal sinus usually empties posteriorly, into one or the other lateral sinus, most commonly the right one. The variations in the arrangement of the vessels at the torcular Herophili will be described in detail later.

In an examination of several hundred skulls, Streit found a number of anomalies in the formation of the superior longitudinal sinus. He found partial duplication of the superior longitudinal sinus, as the result of the presence of a septum in its lumen. This occurred most commonly in its posterior part. In some cases, there was a complete vertical septum, dividing the sinus into a right and left compartment, each compartment ending in the lateral sinus of the corresponding side.

The superior longitudinal sinus may split several centimeters above the internal occipital protuberance. Labbé reported such a case, in which the sinus split 3 cm. above the internal occipital protuberance. The thicker of the two arms joined both lateral sinuses. The thinner arm ended in the right lateral sinus 2 cm. from the torcular.

In a case reported by Malacre, the two branches of the superior longitudinal sinus ran along the lambdoid or occipitoparietal sutures, and emptied into the knees of both sigmoid sinuses. The lateral sinuses were very narrow.

The occurrence of duplication of the superior longitudinal sinus is readily explained by the fact that this sinus is developed from two capillary networks, which lie on either side of the midline, and gradually approach

each other to fuse into a single vessel. If this fusion is interrupted for any reason, there is partial duplication of the sinus. The reason why the duplication is most common at the posterior end of the sinus is because the

FIG. 18. ANOMALY OF SUPERIOR LONGITUDINAL SINUS. SUPERIOR LONGITUDINAL SINUS PASSES DOWN TO RIGHT JUGULAR BULB. RIGHT LATERAL SINUS IS VERY SMALL. OCCIPITAL SINUS IS DOUBLE, AND EACH EMPTIES INTO VERTEBRAL VEINS. (*After Streit.*)

CS, cavernous sinus; IPS, inferior petrosal sinus; JB, jugular bulb; LS, lateral sinus; OS, occipital sinus; SLS, superior longitudinal sinus; SPS, superior petrosal sinus.

fusion starts at the anterior end of the sinus, and gradually works its way backward.

Portal reported 2 cases of complete absence of the superior longitudinal sinus.

The superior longitudinal sinus may be very small, and the inferior longitudinal sinus may be large.

Knott reported 1 case in which the superior longitudinal sinus emptied directly into the straight sinus, instead of into one or the other of the lateral sinuses.

Streit described 1 case in which the superior longitudinal sinus passed across the posterior cranial fossa and emptied into the jugular bulb (Fig. 18). After taking its normal course to a point above the internal occipital protuberance, it crossed to the right of this protuberance, and perforating the tentorium cerebelli, passed in a straight line obliquely to the right and downward, entering the right jugular bulb. The straight sinus emptied into the torcular Herophili. The left lateral sinus was normal. The sigmoid portion of the right lateral sinus was narrower than usual, while the horizontal portion was extremely narrow. There were two posterior occipital sinuses, both of which originated from the torcular Herophili, and passed along the margin of the foramen magnum, emptying into the vertebral veins. There was no connection between the occipital sinuses and the bulb. The right occipital sinus communicated in several places with the extension of the superior longitudinal sinus. There was a communication between the superior longitudinal sinus and the torcular Herophili, and a communication between the jugular bulb and the vertebral veins.

The Inferior Longitudinal Sinus. The inferior longitudinal sinus is a sickle-shaped vessel, which runs parallel to the superior longitudinal sinus, along the lower or free margin of the falx cerebri. It terminates posteriorly in the straight sinus, at the anterior margin of the tentorium cerebelli. At this point it is joined by the vena magna Galeni. It receives branches from the falx cerebri, and sometimes from the medial surfaces of the cerebral hemispheres. The inferior longitudinal sinus is circular on cross

section, and is comparatively small. Occasionally, when the superior longitudinal sinus is small, the inferior longitudinal sinus is very large.

The Straight Sinus. The straight sinus or sinus rectus is formed

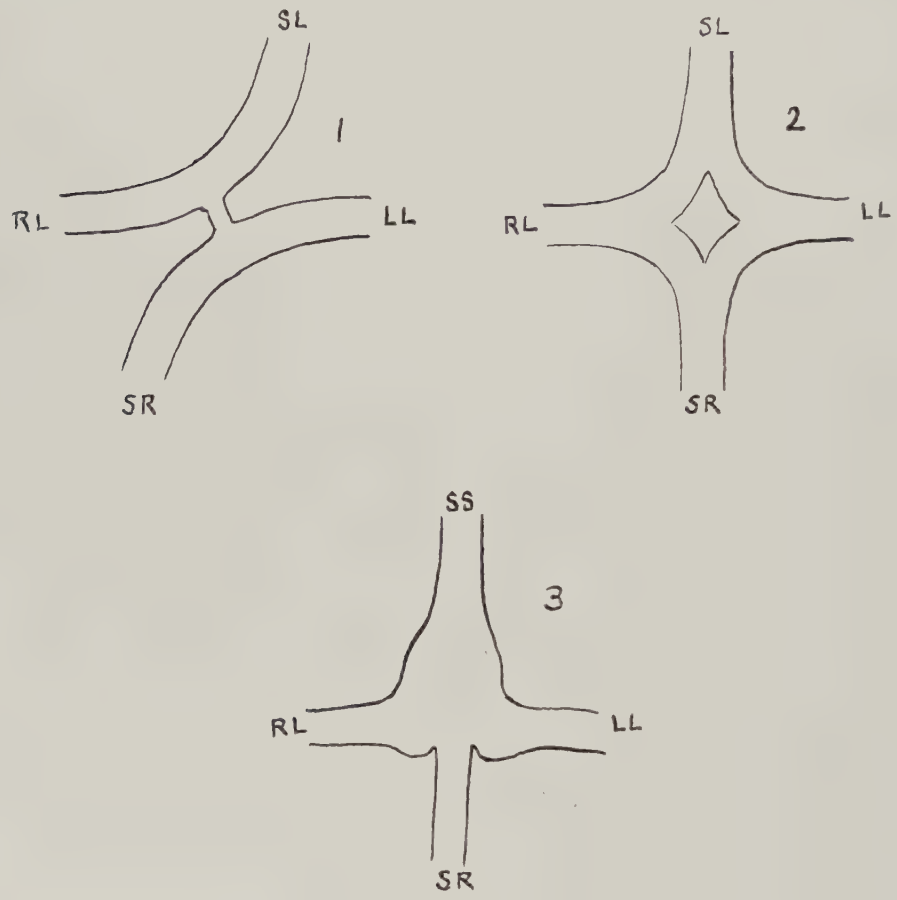

FIG. 19. VARIETIES OF ARRANGEMENT OF SINUSES AT TORCULAR HEROPHILI. (*After Poirier.*)

LL, left lateral sinus; SR, straight sinus; RL, right lateral sinus; SL, superior longitudinal sinus.

by the junction of the inferior longitudinal sinus with the vena magna Galeni. It lies on the tentorium cerebelli along the line of attachment of the falx cerebri to the tentorium. It usually ends posteriorly in the left lateral

sinus. It is a small straight vessel about 2 inches in length. It receives a few branches from the tentorium, and some superior cerebellar veins.

The superior longitudinal sinus drains the blood from the surface of the brain, and the straight sinus drains the blood from the central nuclei and the deep portions of the brain.

Torcular Herophili. According to Poirier, the method of branching of the sinuses at the torcular Herophili follows three principal types. In 50 per cent of the cases, the superior longitudinal sinus continues into the right lateral sinus, and the straight sinus continues into the left lateral sinus. There may be a small branch of communication between the two lateral sinuses (Fig. 19, No. 1).

In 30 per cent of the cases, the vessels are united around an islet of connective tissue. The superior longitudinal sinus divides into two branches, and the straight sinus divides into two branches. The right branch of the superior longitudinal sinus and the right branch of the straight sinus unite to form the right lateral sinus. The left branch of the superior longitudinal sinus and the left branch of the straight sinus unite to form the left lateral sinus (Fig. 19, No. 2).

In 20 per cent of the cases, the four sinuses are fused into a common reservoir. This is a true confluens sinuum (Fig. 19, No. 3).

Henrici and Kikuchi examined the torcular region in 35 heads. They removed a portion of the dura and tentorium cerebelli in the region of the torcular, large enough to examine 5 to 8 cm. of the vessels on each side of the torcular. They arrived at the following conclusions:

In 43 per cent of the cases (Fig. 20), the superior longitudinal sinus and the straight sinus divided into two branches, each. One branch of the superior longitudinal sinus joined one branch of the straight sinus to form the lateral sinus of that side. Between these branches is an angular piece of dura and tentorium, which separates the two lateral sinuses from

each other. The right branch of the superior longitudinal sinus is usually larger than the left, while the left branch of the straight sinus is usually larger than the right. The right lateral sinus is usually larger than the left.

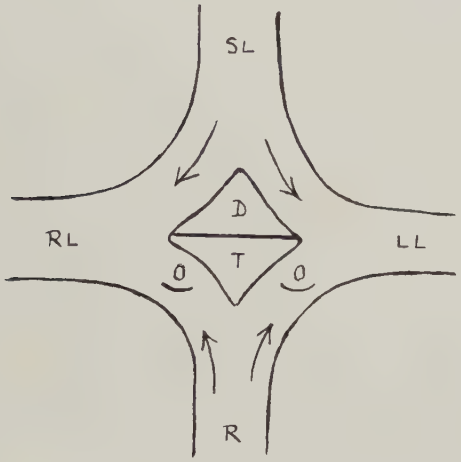

FIG. 20. ARRANGEMENT OF SINUSES AT TORCULAR HEROPHILI, TYPE 1. (*After Henrici and Kikuchi.*)

D, dura of middle fossa, LL, left lateral sinus; O, occipital sinus; R, sinus rectus; RL, right lateral sinus; SL, superior longitudinal sinus; T, tentorium cerebelli.

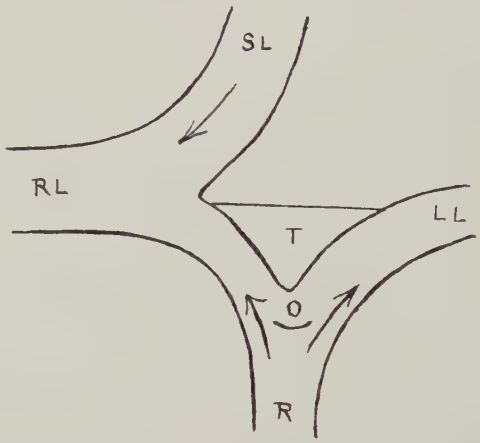

FIG. 21. ARRANGEMENT OF SINUSES AT TORCULAR HEROPHILI, TYPE 2. (*After Henrici and Kikuchi.*)

LL, left lateral sinus; O, occipital sinus; R, sinus rectus; RL, right lateral sinus; SL, superior longitudinal sinus; T, tentorium.

If, in such a case, there is a thrombus in one lateral sinus, the blood from the superior longitudinal sinus and the straight sinus will pass through the arms which lead to the opposite lateral sinus.

In 23 per cent of the cases, the following arrangement was found (Fig. 21): The superior longitudinal sinus was continuous with the right lateral sinus, after joining the right branch of the straight sinus. The straight sinus divided into two branches, and the left lateral sinus was a continuation of the left branch of the straight sinus. The left lateral sinus was much smaller than the right. A small process of dura and tentorium separated the two lateral sinuses.

If, in such a case, there is a thrombus in the left lateral sinus, the blood from the superior longitudinal sinus flows through the right lateral sinus, and the blood from the straight sinus flows through its right arm into

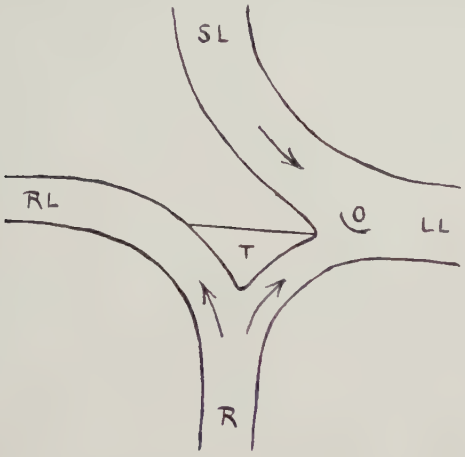

FIG. 22. ARRANGEMENT OF SINUSES AT TORCULAR HEROPHILI, TYPE 3. (*After Henrici and Kikuchi.*)

LL, left lateral sinus; O, occipital sinus; R, sinus rectus; RL, right lateral sinus; SL, superior longitudinal sinus; T, tentorium.

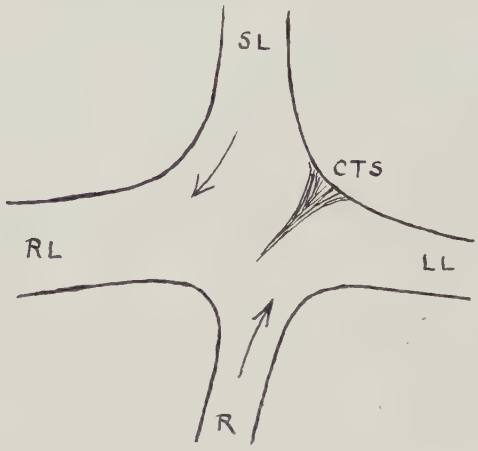

FIG. 23. ARRANGEMENT OF SINUSES AT TORCULAR HEROPHILI, TYPE 4. (*After Henrici and Kikuchi.*)

CTS, connective tissue septum; LL, left lateral sinus; R, sinus rectus; RL, right lateral sinus; SL, superior longitudinal sinus.

the right lateral sinus. If there is a thrombus in the right lateral sinus, the blood from the straight sinus flows through its left arm into the left lateral sinus, and the blood from the superior longitudinal sinus passes through the right and left branches of the straight sinus to the left lateral sinus. If the thrombus in the right lateral sinus extended past the point of entrance of the right branch of the straight sinus, there would be no outflow from the superior longitudinal sinus.

In 11 per cent of the cases, the conditions were the reverse of the previous type (Fig. 22).

In a few of the first three groups of cases, a small canal ran through the dural island, forming a direct connection between the two lateral sinuses. It was so small, however, that it could have no pathological significance.

In 11 per cent of the cases, there was a real confluens sinuum, that is, all four sinuses met in a common reservoir (Fig. 23); but the blood stream from the superior longitudinal sinus did not directly meet that from the straight

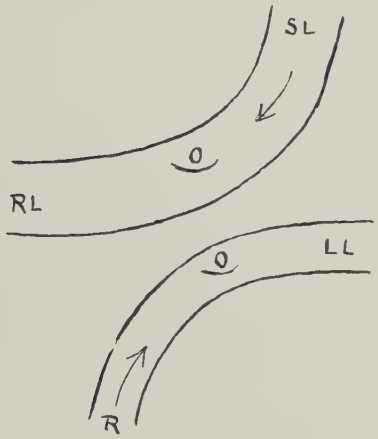

FIG. 24. ARRANGEMENT OF SINUSES AT TORCULAR HEROPHILI, TYPE 5. (*After Henrici and Kikuchi.*)

LL, left lateral sinus; O, occipital sinus; R, sinus rectus; RL, right lateral sinus; SL, superior longitudinal sinus.

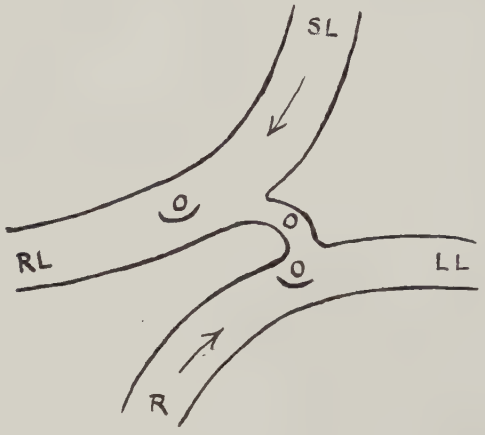

FIG. 25. ARRANGEMENT OF SINUSES AT TORCULAR HEROPHILI, TYPE 6. (*After Henrici and Kikuchi.*)

LL, left lateral sinus; O, occipital sinus; R, sinus rectus; RL, right lateral sinus; SL, superior longitudinal sinus.

sinus. The superior longitudinal sinus bends toward the right, and the straight sinus bends toward the left. The two blood streams pass each other. Sometimes a partial septum was present to indicate a separation of the two streams.

In this class of cases it is comparatively easy for a thrombus to extend from one lateral sinus to the other.

If there is a thrombus in one lateral sinus, blood will pass directly from the superior longitudinal sinus and the straight sinus into the other lateral sinus.

In 1 case, there was complete separation of both lateral sinuses (Fig. 24). The superior longitudinal sinus continued into the right lateral sinus, and the straight sinus continued into the left lateral sinus. If, in this type of

case, there is a thrombus in the right lateral sinus, there is venous stasis in the superficial part of the brain. If there is a thrombus in the left lateral sinus, there is venous stasis in the central parts of the brain. However, if the

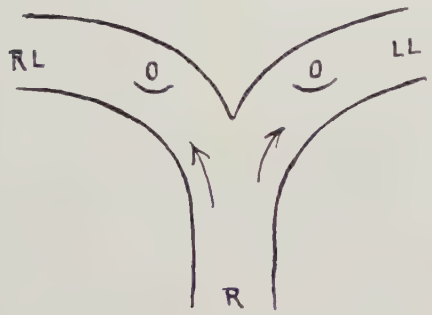

FIG. 26. ARRANGEMENT OF SINUSES AT TORCULAR HEROPHILI, TYPE 7. (*After Henrici and Kikuchi.*)

LL, left lateral sinus; O, occipital sinus; R, sinus rectus; RL, right lateral sinus.

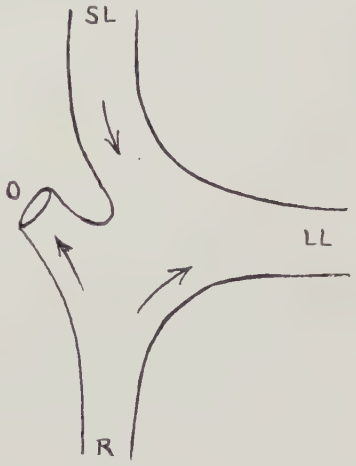

FIG. 27. ARRANGEMENT OF SINUSES AT TORCULAR HEROPHILI, TYPE 8. (*After Henrici and Kikuchi.*)

LL, left lateral sinus; O, occipital sinus; R, sinus rectus; SL, superior longitudinal sinus.

thrombus does not reach as far as the entrance of the occipital sinus into the lateral sinus, the occipital sinus will carry off the blood.

In 1 case, a branch of the occipital sinus connected the two lateral sinuses (Fig. 25).

In 1 case, there was a complete absence of the superior longitudinal sinus (Fig. 26). The straight sinus was very large, and divided into the two lateral sinuses. The occipital sinuses were also large.

In 1 case, the right lateral sinus was absent (Fig. 27). The superior longitudinal sinus continued into the left lateral sinus. The straight sinus divided into two branches, of which the left joined the superior longitudinal sinus to form the left lateral sinus, while the right branch continued as the occipital sinus.

Sinuses at Base of Skull

The Lateral Sinus. There are two lateral sinuses, one on either side of the skull. The vessel has a very tortuous course, starting at the internal occipital protuberance, and ending at the jugular foramen, where it is continued into the jugular bulb. It lies against the inner surfaces of the occipital, parietal and temporal bones.

Each lateral sinus is ordinarily described as consisting of two portions, the horizontal portion and the sigmoid portion. The horizontal portion extends from the internal occipital protuberance to the posteroinferior angle of the parietal bone. It is situated along the line of attachment of the tentorium cerebelli to the occipital and parietal bones. The sigmoid portion of the lateral sinus extends from the posteroinferior angle of the parietal bone to the jugular bulb. It lies in the angle formed by the union of the petrous with the mastoid portion of the temporal bone.

The horizontal portion is from 2 to 2½ inches long. It is slightly curved, the convexity of the curve being directed upward.

The sigmoid portion is from 1½ to 2 inches long. It is s-shaped, and may be roughly divided into a vertical portion and a horizontal portion. There is a bend at the upper end of the vertical portion of the sigmoid sinus. This bend is called the knee of the sinus. The convexity of this bend is directed forward, and it is the point at which the superior petrosal sinus enters the lateral sinus. The vertical limb of the sigmoid sinus is not really vertical, but is directed obliquely from behind forward and inward. This portion of the sinus is from 1 to 1¼ inches in length. It is the portion of the sinus most commonly involved in sinus thrombosis, as it is in close relation to the mastoid antrum. At the lower end of the vertical limb, the sinus curves sharply forward into the horizontal limb of the sigmoid sinus. The horizontal limb is very short. It is really part of a short arc with its concavity directed upward. The end of this arc curves upward and forward, terminating in the jugular bulb.

The width of the lateral sinus varies in different individuals from ¾ to ¼ inch (19.5 to 6.5 mm.). It also varies in width in different parts of its course. The knee, the lower end of the vertical limb and the horizontal

FIG. 28. RELATION OF LATERAL SINUS TO OUTER SURFACE OF SKULL.

Dotted line A passes from external occipital protuberance through asterion and upper margin of external auditory canal. Its posterior two-thirds mark location of horizontal portion of lateral sinus. Dotted line B passes from parietal incisure of temporal bone to tip of mastoid. Marks location of vertical limb of sigmoid sinus.

portion of the lateral sinus are usually the widest portions. The right sinus is ordinarily wider than the left, because the right sinus is usually the continuation of the superior longitudinal sinus, and the left lateral sinus is the continuation of the straight sinus. In the cases in which the left lateral sinus is the continuation of the superior longitudinal sinus, the left lateral sinus is wider than the right. The wider sinus is usually situated more superficially and further forward than the other.

The horizontal portion of the lateral sinus is triangular on cross section, with its convex base directed outward.

The sigmoid portion is semicircular on cross section, the convex surface

FIG. 29. HORIZONTAL SECTION OF TEMPORAL BONE, SHOWING SUPERFICIALLY LOCATED GROOVE FOR SIGMOID SINUS. DISTANCE FROM OUTER SURFACE OF MASTOID TO SIGMOID SINUS IS 1/16 INCH.

FIG. 30. HORIZONTAL SECTION OF TEMPORAL BONE, SHOWING DEEPLY SITUATED GROOVE FOR SIGMOID SINUS. DISTANCE FROM OUTER SURFACE OF MASTOID TO SIGMOID SINUS IS 3/4 INCH.

corresponding to the shape of the groove in the skull in which it lies. The flat surface lies on the surface of the brain.

The horizontal portion of the lateral sinus corresponds to the posterior two-thirds of a line drawn on the outer surface of the skull, extending from

the external occipital protuberance through the base of the mastoid process to the external auditory canal (Fig. 28).

The vertical limb of the sigmoid sinus corresponds to a line on the outer surface of the skull, extending from the parietal incisure of the temporal bone to the tip of the mastoid process. The knee of the sinus is usually level with the upper margin of the external auditory canal, and is situated from 1 to 25 mm. behind the canal. In wide mastoids, the knee of the sinus is usually a considerable distance behind the external auditory canal. In narrow mastoids, the sinus is very close to the canal. When the knee is situated far forward, it is usually superficial, and in extreme cases it is external to the mastoid antrum. An attempt to reach the antrum in such cases is fraught with considerable danger of injury to the sinus. The distance between the sigmoid sinus and the surface of the mastoid varies from 2.6 mm. to 1.2 cm. (Figs. 29 and 30).

The more oblique the vertical limb of the sigmoid sinus, the further removed is the knee of the sinus from the external auditory canal. According to Ballance, a long skull with a long occiput has an oblique vertical limb of the sigmoid sinus, and a short skull with a short occiput has a more upright vertical limb. The more upright the vertical limb, the sharper is the bend from the sigmoid sinus to the jugular bulb.

Prentiss believes that the position and the obliquity of the vertical limb of the sinus can be determined by the position of the supramastoid crest, that is, the posterior root of the zygoma. The supramastoid crest is developed along the lateral suture line between the squamous portion of the temporal bone and the petromastoid bone. This suture line and the supramastoid crest determine the position of the superior surface of the petrous pyramid. If the supramastoid crest runs obliquely upward and backward, it indicates that the upper surface of the petrous pyramid approximates the vertical rather than the horizontal plane, and therefore the same is true of

the posterior surface of the petrous pyramid. If the supramastoid crest is more horizontal, it indicates that the superior and posterior surfaces of the petrous pyramid approximate the horizontal plane.

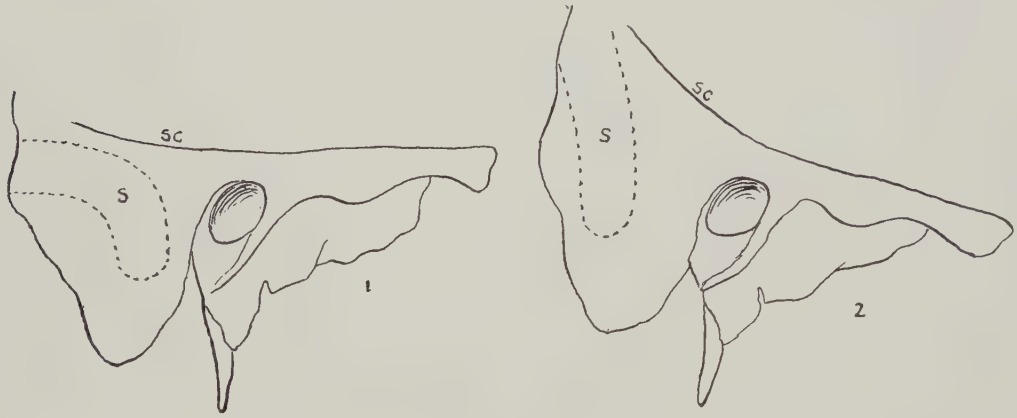

FIG. 31. 1. SUPRAMEATAL CREST (SC) HORIZONTAL. VERTICAL LIMB OF SIGMOID SINUS (S) ENCROACHES ON EXTERNAL AUDITORY CANAL, AND BEND OF KNEE IS SHARP.

2. SUPRAMEATAL CREST OBLIQUE. VERTICAL LIMB OF SIGMOID SINUS DOES NOT ENCROACH ON EXTERNAL AUDITORY CANAL, AND BEND OF KNEE IS NOT SHARP. (*After Prentiss.*)

FIG. 32. 1. EXTERNAL AUDITORY CANAL (C) OBLIQUE. SINUS (S) PARALLEL TO CANAL, AND SITUATED FAR FROM CANAL.

2. EXTERNAL AUDITORY CANAL AT RIGHT ANGLES TO SURFACE OF SKULL. SINUS APPROACHES CANAL CLOSELY. (*After Prentiss.*)

In the first class of cases, the lateral sinus, in making its bend at the knee, encroaches very slightly on the mastoid antrum and external auditory canal, on account of the highly placed posterior surface of the petrous pyramid. In the second class of cases, the sinus must encroach markedly on

the bone to make the bend, in order to keep the lumen of the sinus intact. In other words, in the first class, the bend of the knee is less sharp than in the second class (Fig. 31).

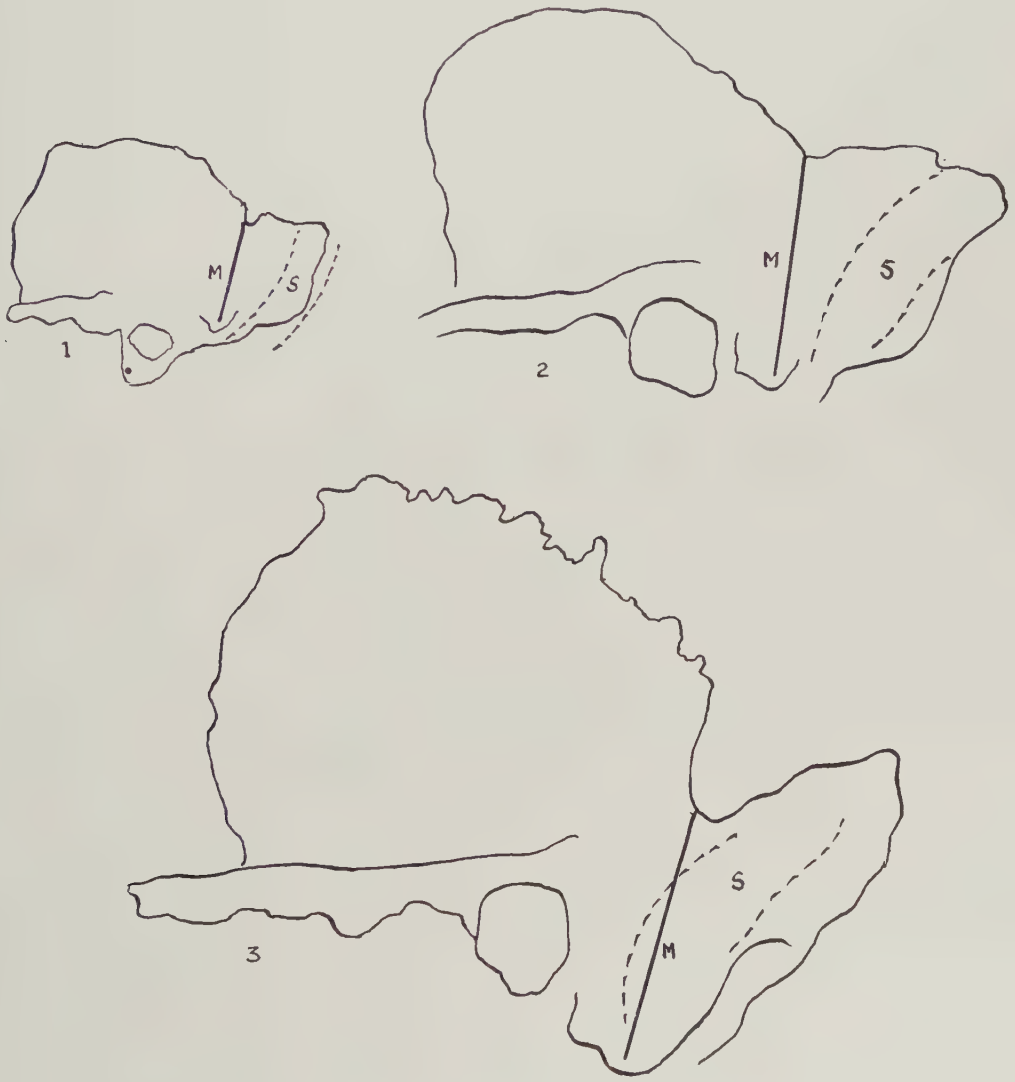

FIG. 33. POSITION OF SIGMOID SINUS WITH REFERENCE TO MACEWEN'S LINE AT VARIOUS AGES. (*After P. Rudloff.*)

M, Macewen's line; S, sigmoid sinus. 1. Temporal bone at birth. 2. Temporal bone at six years of age. 3. Temporal bone of adult.

If the external auditory canal is very oblique, it parallels the approach of the sinus, and the sinus keeps away from the canal. If the canal is at right angles to the surface of the skull, the sinus approaches the canal more closely (Fig. 32).

The line which is drawn from the lowest point of the parietal incisure of the temporal bone to the tip of the mastoid process is called Macewen's line. In the adult this line marks the midline of the vertical limb of the sigmoid sinus. But Rudloff found that this rule does not hold true in children. In young children Macewen's line lies a considerable distance in front of the vertical limb of the sigmoid sinus. With increasing age, the sinus comes to lie further forward, and in the adult, the line passes through the center of the vertical limb (Fig. 33).

The upper part, or the knee, is the most superficial portion of the sigmoid sinus. The lower end lies furthest from the surface of the skull. For this reason, it is safest, in removing the superficial mastoid cells, to begin at the lower part of the mastoid and work upward, as there is less likelihood of injuring the sinus in this way.

The vertical limb of the sigmoid sinus lies between the mastoid process and the cerebellum, the cerebellum lying to the inner side of the sinus (Fig. 34). The cerebellar dura, when it reaches the angle between the mastoid process and the petrous pyramid, splits into two layers, which form the outer and inner, or the parietal and visceral walls of the sinus. The two layers reunite in front of the sinus, at the posterior surface of the petrous pyramid. In the visceral wall of the sinus, about midway between the knee and the lower extremity of the vertical limb, is situated the saccus endolymphaticus. A labyrinthine suppuration which extends into the saccus endolymphaticus sometimes gives rise to an infection of the sigmoid sinus.

The mastoid antrum is situated in front of and somewhat external to this portion of the sinus (Figs. 34 and 35). When the vertical limb of the

sinus is very superficial and situated far forward, however, the antrum may be internal to the sinus.

The knee of the sinus lies behind and external to the facial nerve as the

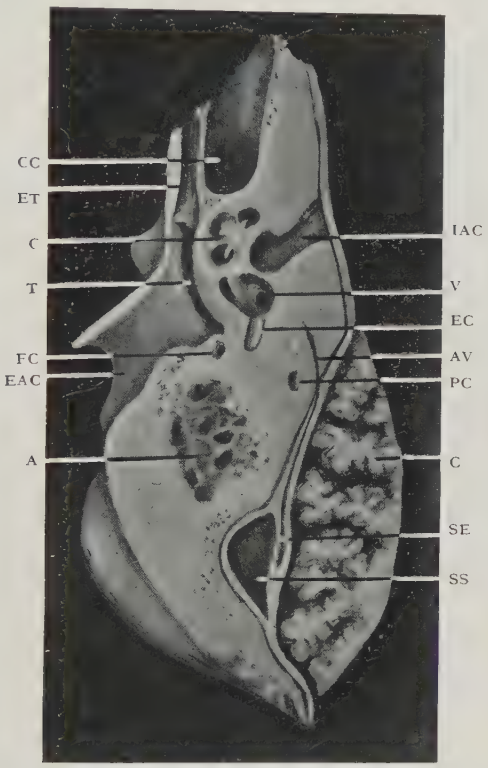

FIG. 34. HORIZONTAL SECTION OF LEFT TEMPORAL BONE, SIGMOID SINUS AND PORTION OF CEREBELLUM, VIEWED FROM ABOVE.

A, antrum; AV, aqueductus vestibuli; C, cochlea and cerebellum; CC, carotid canal; EC, external semicircular canal; ET, bony Eustachian tube; EAC, external auditory canal; FC, Fallopian canal; IAC, internal auditory canal; PC, posterior semicircular canal; SE, saccus endolymphaticus; SS, sigmoid sinus; T, tympanic cavity; V, vestibule.

latter curves around the oval window (Figs. 36 and 37). As the nerve and sinus pass downward, they approach each other anteroposteriorly and laterally. In the lower part of their course, they cross each other. At the point where the facial nerve makes its exit from the temporal bone, that is, at the stylomastoid foramen, the sinus is situated directly internal to the

nerve (Fig. 38). This is the point at which the sinus enters the jugular bulb. If one were to try to expose the sinus at this point by working directly inward through the mastoid, the facial nerve would be inevitably injured.

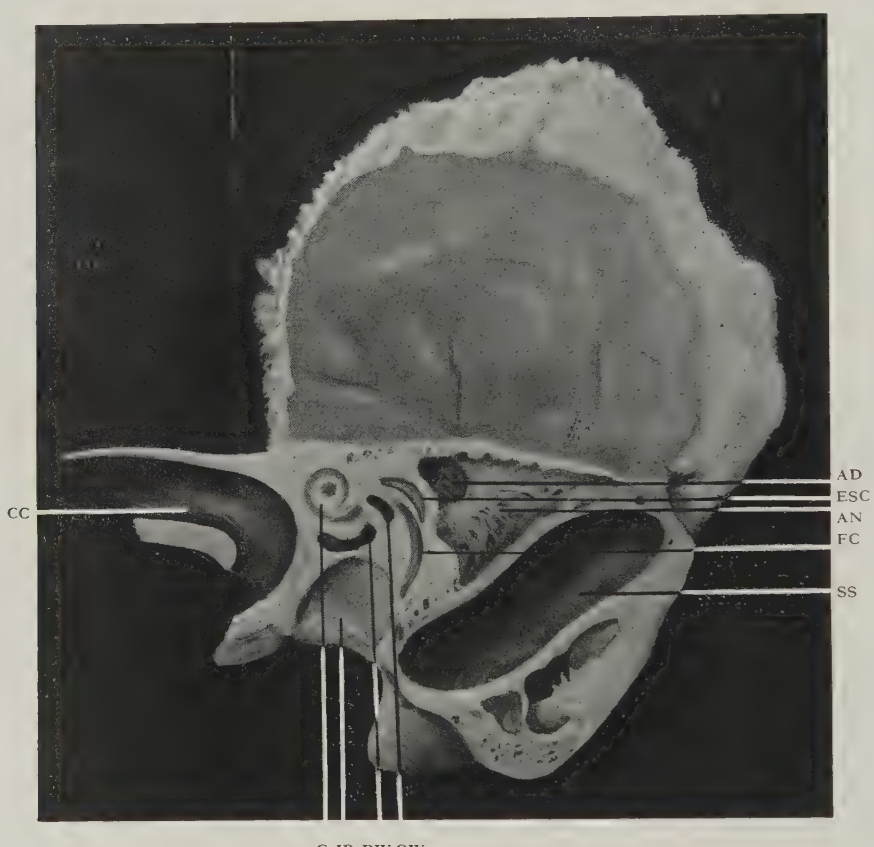

FIG. 35. RIGHT TEMPORAL BONE. VERTICAL SECTION PARALLEL TO POSTERIOR SURFACE OF PETROUS PYRAMID, VIEWED FROM WITHIN OUTWARD.

AD, aditus; AN, antrum; C, cochlea; CC, carotid canal; ESC, external semicircular canal; FC, Fallopian canal; JB, jugular bulb; OW, oval window; RW, round window; SS, sigmoid sinus.

In order to avoid injury to the nerve, the operator must first remove the bone posterior to the nerve, and when a sufficiently deep level has been reached he must remove the bone internal to the nerve, until the sinus is uncovered. In other words, the work must be done around a corner.

In uncovering the cerebellum by removing the mastoid process, it will be seen that the vertical limb of the sigmoid sinus passes diagonally across the exposed portion of the cerebellar dura from behind and above, in a

FIG. 36. RELATION OF FACIAL NERVE TO SIGMOID SINUS IN CORONAL SECTION.
E, external side; F, facial nerve; I, internal side; JB, jugular bulb; SS, sigmoid sinus.

direction downward and forward. The sinus divides this portion of the cerebellar dura into two triangles, one of which lies in front of and above the sinus, and is known as Trautmann's triangle, while the other lies behind and below the sinus (Fig. 39). Trautmann's triangle has its base above and its apex below. The base of the triangle is formed by the superior petrosal sinus. The anterior margin of the triangle is formed by the posterior semicircular canal and the posterior margin of the triangle is formed by the anterior border of the vertical limb of the sigmoid sinus. It is through Trautmann's triangle that we usually explore for a cerebellar abscess, as we can most easily reach the median lobe of the cerebellum and the median portion of the lateral

lobe by this route. An abscess in the lateral portion of the lateral cerebellar hemisphere is best reached through the triangle which is situated behind the sigmoid sinus. The vertical limb of the sigmoid sinus contracts the area

FIG. 37. RELATION OF FACIAL NERVE TO SIGMOID SINUS IN SAGITTAL SECTION.
A, anterior; F, facial nerve; JB, jugular bulb; P, posterior; SS, sigmoid sinus.

through which the surgeon can reach a cerebellar abscess, and it also interferes with free drainage of an abscess in the cerebellum. Ballance has advised packing off the sinus above and below the operative area, and dividing the sinus, in order to secure adequate drainage in cases of large cerebellar abscess. The writer employed this method in one case with a very gratifying result.

When the vertical limb of the sigmoid sinus reaches the lower extremity of the mastoid process, the sinus bends sharply forward and then upward, and passes into a dilated reservoir called the jugular bulb. The angle which is formed in the sinus as it bends forward and upward varies considerably

in different heads. When the mastoid is broad and the sinus is situated fairly far backward, the angle is wide. When the mastoid is narrow and the sinus is far forward, the angle is very acute. Between the lower part of the

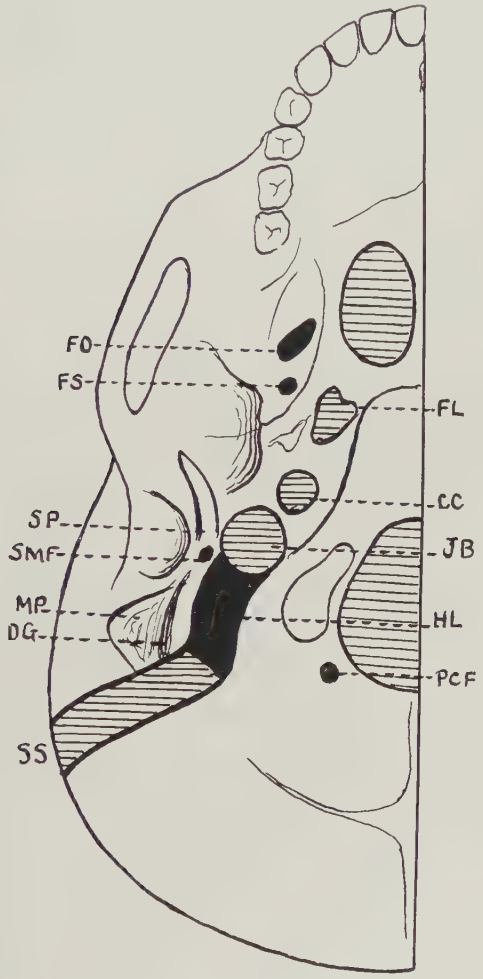

FIG. 38. UNDER SURFACE OF SKULL SHOWING RELATIONS OF HORIZONTAL LIMB OF SIGMOID SINUS.

CC, carotid canal; DG, digastric groove; FL, foramen lacerum; FO, foramen ovale; FS, foramen spinosum; HL, horizontal limb of sigmoid sinus; JB, jugular bulb, MP, mastoid process; PCF, posterior condyloid foramen; SMF, stylomastoid foramen; SP, styloid process; SS, vertical limb of sigmoid sinus.

vertical limb of the sigmoid sinus and the jugular bulb, there is a wedge of bone which is formed by the lower part of the base of the petrous pyramid.

It contains the lower part of the facial nerve. A portion of this wedge of bone must be removed, in order to expose the jugular bulb from behind.

The horizontal limb of the sigmoid sinus is bounded above by the

FIG. 39. CEREBELLAR TRIANGLES.

C, posterior semicircular canal; O, superior petrosal sinus; P, posterior cerebellar triangle; S, sigmoid sinus; T, Trautmann's triangle.

wedge of bone just described. It is bounded below by that portion of the occipital bone which lies in the angle between the posterior margin of the jugular foramen and the groove for the digastric muscle (Fig. 38). It is in relation externally with the facial nerve, just before the exit of the latter from the stylomastoid foramen. It is in relation internally with the under surface of the cerebellar hemisphere. If the mastoid tip is removed, the digastric muscle drawn to the side, and the bone internal to the digastric muscle removed, the lower surface of this portion of the sinus will be exposed. This is done in the Grunert operation on the jugular bulb.

The lateral sinus receives blood from the superior longitudinal sinus, the straight sinus, the superior petrosal sinus, and the occipital sinus. It

empties into the internal jugular vein through the jugular bulb. It receives veins from the upper and lower surfaces of the cerebellum and the posterior part of the cerebellar hemispheres. It also receives some of the diploic veins, and some small veins from the middle ear, antrum and mastoid cells. These latter veins enter the sigmoid sinus mostly on the anterior margin in the neighborhood of the knee.

The right lateral sinus is usually larger than the left, because the former as a rule receives the blood from the superior longitudinal sinus, while the latter is continuous with the straight sinus. The former drains the surface of the brain and the skull while the latter drains the central ganglia of the brain.

The lateral sinus communicates with the vessels on the outer surface of the skull through four emissary veins; the mastoid emissary, the posterior condyloid emissary, the occipital emissary, and sometimes a vein which passes through the anterior condyloid foramen with the hypoglossal nerve.

The mastoid emissary is a vein which usually arises from the posterior margin of the vertical limb of the sigmoid sinus. Its exact location varies. Sometimes it starts from the upper part of the vertical limb, and sometimes from its middle portion. It is directed backward and outward, and makes its exit through the outer surface of the mastoid, near the posterior margin of the latter. It may have a tortuous course in the bone. The external opening may be in the occipital bone, as much as 2 cm. behind the occipitomastoid suture. Sometimes the vessel lies in a groove on the inner surface of the mastoid, before it enters the bone. In this location, it lies in contact with the dura. It may also lie in a groove on the outer surface of the skull. There may be two mastoid foramina on one or both sides. The foramen was wanting on one side in 8 per cent of the skulls examined by Thomson.

The mastoid foramen was missing on the right side in 78 of 429 skulls examined by Streit, and on the left side in 65 skulls. The foramen was less

than 3 mm. in diameter in 238 skulls on the right side, and in 247 skulls on the left side. It was between 3 and 4½ mm. in diameter in 32 skulls on the right side, and in 45 skulls on the left side. It was between 4½ and 6 mm. in 10 skulls on the right side, and in 16 skulls on the left side. In 3 skulls, the canal was 7 mm. in diameter, in 2 skulls it was 9 mm. and in 1 skull it was 10 mm. In the remaining skulls, there were two or more openings on the outer surface, and one opening on the inner surface. The average size was 2 mm.

The mastoid emissary usually ends externally in the occipital vein. The occipital vein empties into the deep cervical vein, which, in turn, ends in the vertebral vein before the latter joins the subclavian. Sometimes the emissary ends in the posterior auricular vein. The latter empties into the external jugular. There may be numerous anastomoses at the exit of the emissary, so that the lateral sinus may communicate with the external, internal and posterior jugular veins. Occasionally the external jugular vein consists almost entirely of a continuation of the emissary.

Otto describes a case in which there was a connection between the mastoid canal and the petrosquamous sinus.

The emissary usually serves to carry blood from within the skull to the outside. But on occasions when the blood pressure within the skull is very low, the emissary may carry blood from without into the interior of the skull. When there is a thrombus in the lower part of the sigmoid sinus, the emissary may serve to carry off a considerable part of the intracranial blood to the outside of the skull.

Thrombi in the sigmoid sinus may extend through the emissary to the veins of the scalp. In perisinous abscess, when there is a collection of pus in the sinus groove, some of the pus may find its way through the canal, by the side of the emissary, and infiltrate the tissues of the scalp at the point of emergence of the emissary.

The occipital emissary vein is an inconstant winding communication between the region of the torcular Herophili and the occipital vein. Its point of exit is at the external occipital protuberance. There may be more than one occipital emissary.

The condyloid emissary passes through the posterior condyloid foramen, and joins the sigmoid sinus to the deep cervical veins and the vertebral plexus.

It runs obliquely from in front, above and laterally, in a direction backward, downward and medially; so that if both canals were prolonged downward and backward, they would meet. Its length varies. In extreme cases it is 2 cm. long. The plate of bone which separates it from the posterior fossa of the skull may be very thin, or even dehiscent. It may be double at one or both ends.

In a number of cases, there is a connection between the posterior and anterior condyloid canals.

According to Thomson, the posterior condyloid canal is missing in 10 per cent of the cases on the right side, in 17 per cent on the left side, and in 4 per cent on both sides. It averages 2 to 2½ mm. in diameter. Its maximum diameter is 9 mm.

Streit examined 368 skulls. In 101 he found that the posterior condyloid canal was missing on the left side, and in 105 he found it was missing on the right side. In 185 skulls the condyloid emissary was less than 2 mm. in diameter on the left side, and in 169 it was less than 2 mm. in diameter on the right side. In 69 it was from 2 to 4 mm. in diameter on the left side and in 62 it was from 2 to 4 mm. in diameter on the right side. In 11 it was from 4 to 6 mm. in diameter on the left side and in 62 it was from 4 to 6 mm. in diameter on the right side. In 3, it was 7 mm. in diameter, and in 3, it was 8 mm. in diameter.

A collection of pus in the posterior cranial fossa may find its way through the posterior condyloid foramen to the outer surface of the skull. This gives

rise to a cellulitis in the upper part of the posterior cervical triangle, in the deep tissues of the neck under the deep cervical fascia.

The anterior condyloid foramen usually contains several small veins,

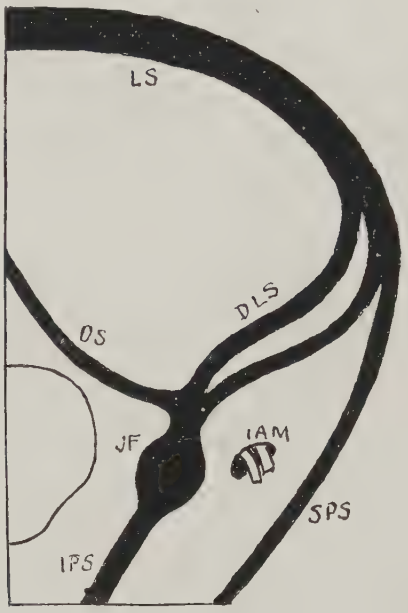

FIG. 40. DOUBLE SIGMOID SINUS. (*After J. M. Brown.*)

DLS, duplicated portion of sigmoid sinus; IAM, internal auditory meatus; IPS, inferior petrosal sinus; LS, lateral sinus; OS, occipital sinus; SPS, superior petrosal sinus.

along with the hypoglossal nerve. The veins come from the dura and pia. Occasionally, the canal is very wide and contains a large vein which joins the sigmoid sinus to the deep veins of the neck. When this occurs the posterior condyloid vein is usually absent.

Variations of the Lateral Sinus. The horizontal portion of the lateral sinus may be partially divided by a horizontal septum into two channels.

Brown reported a case of double sigmoid sinus from the knee to the junction of the occipital sinus with the sigmoid. The two divisions of the sinus were separated from each other by a ridge of bone 8 mm. wide. The length of the duplicated portion of the sinus was 4 cm. (Fig. 40).

Other cases of double lateral sinus have been reported by Knott, Sperino, Streit, Hallett, Meckle, Dumont, Heine and Beyer.

A double lateral sinus may be explained by the persistence of the

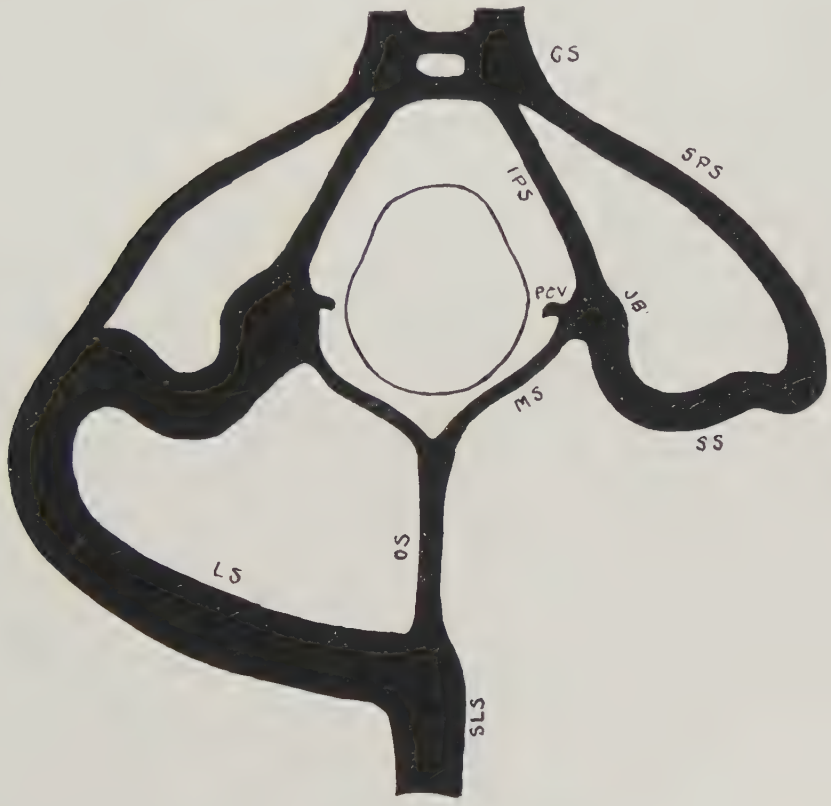

FIG. 41. LEFT LATERAL SINUS PRESENT ONLY FROM ENTRANCE OF SUPERIOR PETROSAL SINUS TO JUGULAR BULB.

CS, cavernous sinus; IPS, inferior petrosal sinus; JB, jugular bulb; LS, lateral sinus; MS, marginal sinus; OS, occipital sinus; PCV, posterior condylar vein; SLS, superior longitudinal sinus; SPS, superior petrosal sinus; SS, sigmoid sinus.

middle portion of the primary head vein, after the lateral sinus is developed dorsal to the otic vesicle. The lateral sinus forms one of the channels of the double sinus, and the middle portion of the primary head vein forms the other channel. It may also be explained by the fact that the capillary network

from which the lateral sinus is developed may fuse into two channels instead of one.

In some of the skulls, the lower end of the sigmoid sulcus, just before its

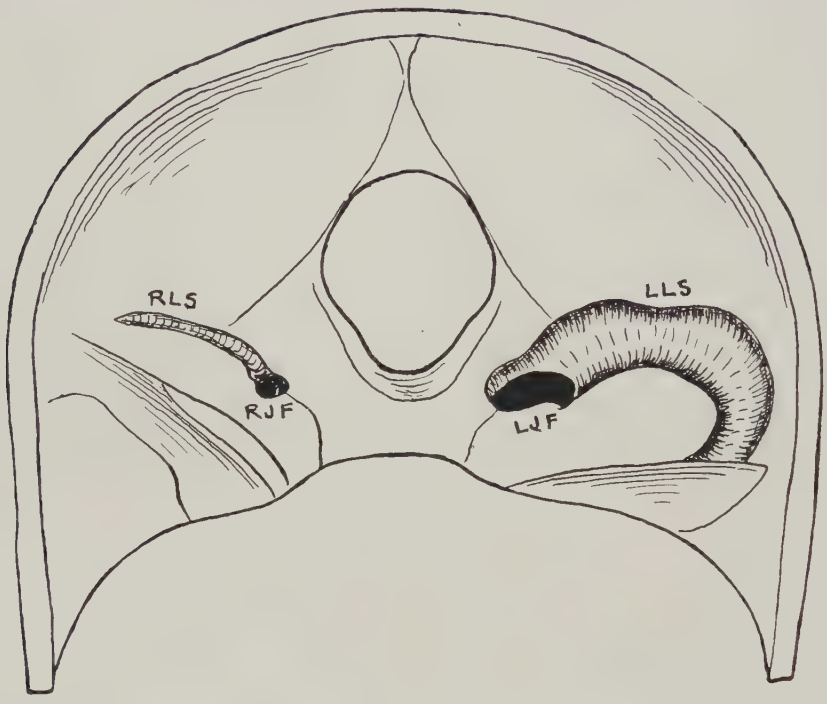

FIG. 42. SKULL SHOWING VERY NARROW GROOVE FOR RIGHT SIGMOID SINUS, AND VERY SMALL RIGHT JUGULAR FORAMEN. (*After Rohrbach.*)

LJF, left jugular foramen; LLS, left sigmoid sinus; RJF, right jugular foramen; RLS, right sigmoid sinus.

entrance into the bulb, is changed into a canal. The horizontal portion of the lateral sinus may be absent, and the sigmoid portion may be present from the mouth of the superior petrosal sinus downward (Fig. 41).

The horizontal portion of the lateral sinus may be very narrow. Knott found a case in which the right lateral sinus was 1.5 mm. in diameter, and passed out of the skull through an enlarged mastoid foramen. Malacre described a skull in which the horizontal portions of both lateral sinuses were normal in size, but both passed out of the skull through enlarged mastoid foramina. The right mastoid foramen was 10 mm. wide.

Budde described a case in which both jugular foramina were very small, and the right mastoid foramen was 1 cm. wide. The mastoid foramen assumed the function of the jugular foramen in these cases.

Rohrbach reported a case of metastatic carcinoma of the left side of the neck. In the course of the operation, the internal jugular vein was ligated. The operation was immediately followed by coma, with death six days later. Post-mortem examination showed circulatory disturbances in the brain, due to an incomplete development of the right lateral sinus, and the right internal jugular vein. The sinus was only large enough to admit a small probe (Fig. 42).

The bony covering of the sigmoid sinus may be very thin. This is especially likely to occur at the knee of the sinus, and at the end of the horizontal limb just before it bends upward into the bulb.

Zuckerkandl found the sigmoid fossa so deep in 1 case, that it occupied the entire jugular process of the occipital bone. The mastoid process may be a thin shell of bone enveloping the sinus. There may be a dehiscence over the sinus. Zuckerkandl, Hartmann, Hyrtl and Brückner described cases with a dehiscence over the sinus.

The posterior canal wall may be so thin as to constitute a transparent plate of bone over the sinus knee. There may be a dehiscence over the lower part of the sigmoid sinus, near the mastoid groove internal to the styloid process.

The Jugular Bulb. This is the connecting link between the sigmoid sinus and the internal jugular vein. It is a dilated reservoir which lies under the floor of the middle ear.

The sigmoid sinus opens into the bulb from behind, and the bulb is continuous at its lower extremity with the internal jugular vein. In order to reach the jugular vein, the bulb passes through the posteroexternal compartment of the jugular foramen. The upper extremity of the bulb is

rounded and is called the dome of the jugular bulb. It lies in the jugular fossa, which is a rounded cavity hollowed out of the under surface of the petrous pyramid.

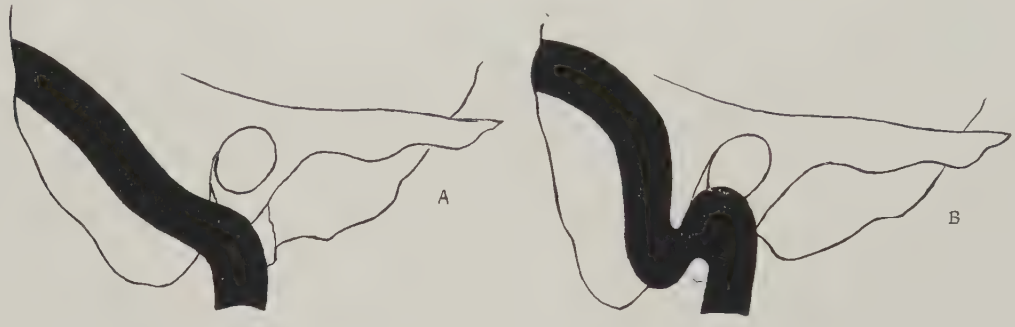

FIG. 43. A. SIGMOID SINUS SITUATED FAR BACK, AND JUGULAR BULB ALMOST DIRECT CONTINUATION OF LINE OF SIGMOID SINUS. DOME OF JUGULAR BULB LOW. B. SIGMOID SINUS FAR FORWARD, AND SHARP BEND UPWARD INTO JUGULAR BULB. DOME OF JUGULAR BULB HIGH.

The size of the jugular bulb varies very much in different individuals. In the average case, it is about 15 mm. wide and 20 mm. high. It may be very narrow with a low dome, or it may be very wide with a high dome. The top of the dome, is, in some cases, as much as ¾ of an inch above the level of the horizontal limb of the sigmoid sinus. In other cases it is not more than ¼ of an inch higher than the horizontal limb.

When the sigmoid sinus is situated far back in the mastoid, the jugular bulb is almost a direct continuation of the line of the vertical limb of the sigmoid sinus, and the dome of the bulb is low. When the sigmoid sinus is far forward, it bends sharply upward into the jugular bulb. In these cases the jugular fossa is very deep, with a high dome (Fig. 43).

The jugular bulb lies under the floor of the middle ear (Fig. 44). When the bulb is very small, it is separated from the floor of the middle ear by a comparatively thick layer of bone. This layer is sometimes 1 cm. thick. It may consist of compact bone, but it frequently contains a few cells. The tympanic nerve, vein and artery pass through this layer of bone. Infection

may travel along these canals from the middle ear to the jugular bulb. The cells in this layer of bone may become diseased, and transmit infection to the bulb.

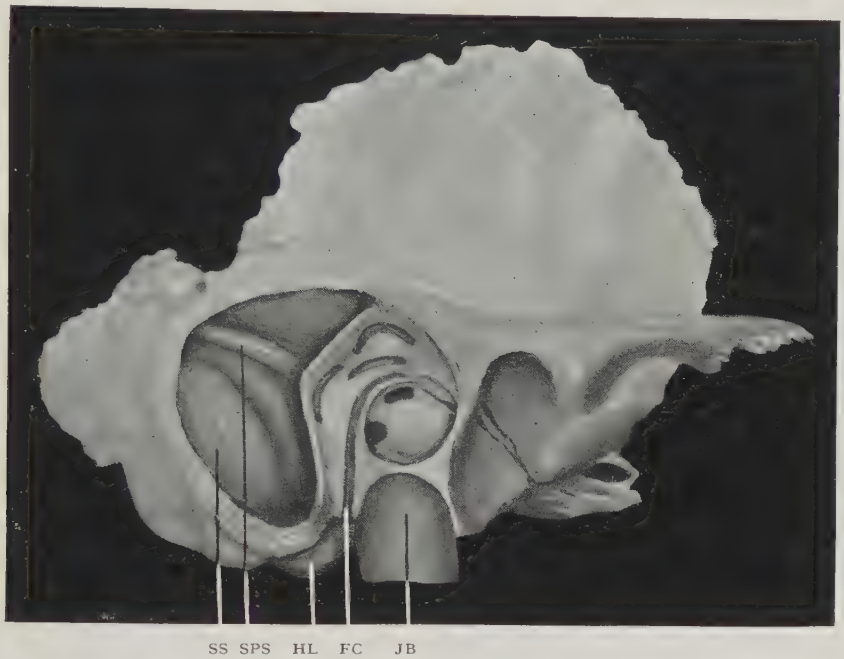

FIG. 44. RIGHT TEMPORAL BONE. RADICAL MASTOID OPERATION, SEMICIRCULAR CANALS AND FALLOPIAN CANAL OPENED, INNER TABLE OVER MIDDLE AND POSTERIOR CRANIAL FOSSAE REMOVED.

FC, Fallopian canal; HL, horizontal limb of sigmoid sinus; JB, jugular bulb; SPC, superior petrosal sinus; SS, sigmoid sinus.

When the bulb is very large, it pushes its way up into the middle ear. In these cases, the floor of the middle ear is very thin. There may even be a dehiscence in the floor. When the dome of the jugular bulb is high, the floor of the middle ear bulges upward. The bulb may reach almost to the middle of the vertical limb of the sigmoid sinus, and the cochlea may be pushed back by it. Zaufal reported a case in which the dome of the jugular bulb shut off the round window.

There may be a dehiscence not only in the floor of the middle ear, but also in the posterior surface of the petrous pyramid, in the layer of bone which separates the jugular fossa from the spinal canal, or in the base of

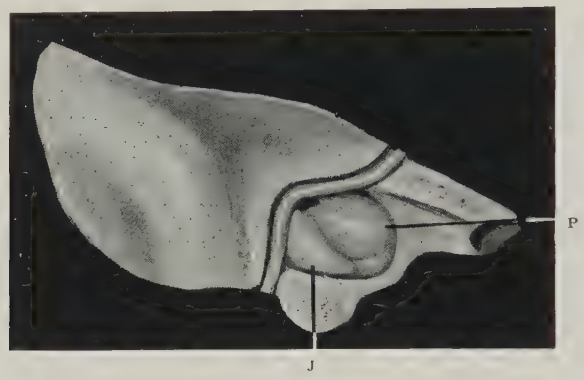

FIG. 45. INNER TYMPANIC WALL, SHOWING VERY HIGH JUGULAR FOSSA. (*After Beyer.*)

J, roof of jugular fossa; P, promontory.

the skull, between the mastoid groove and the posterior part of the condyloid process.

Beyer described a temporal bone in which the inner tympanic wall showed a marked prominence, divided by an oblique furrow into a smaller posterior and a larger anterior portion. Jacobson's nerve ran obliquely across this prominence. The posterior portion of the prominence was triangular, extending upward and backward. It took in the width of the niche of the round window. It had an irregular surface and filled the sinus tympani. The promontory was pushed forward. The oval window was horizontal. The posterior portion of the prominence on the inner tympanic wall was the roof of the jugular fossa (Fig. 45). The sigmoid sulcus was also very large and deep. The jugular fossa extended laterally to the stylomastoid foramen. It extended upward under the posterior canal wall. As a result of the displacement laterally and backward, it came into close relation to the facial nerve. As a

result of extension of the fossa forward, the wall between it and the carotid canal was reduced to a very thin plate of bone.

When the jugular bulb pushes up the floor of the middle ear, and there

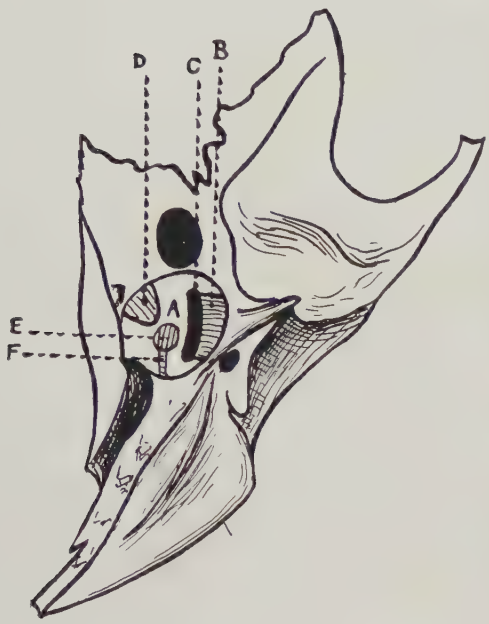

FIG. 46. UNDER SURFACE OF PETROUS PYRAMID.

A, jugular fossa. Shaded areas in jugular fossa show projections of various structures which lie above jugular fossa upon latter; B, external auditory canal; C, middle ear; D, internal auditory canal; E, vestibule; F, posterior semicircular canal.

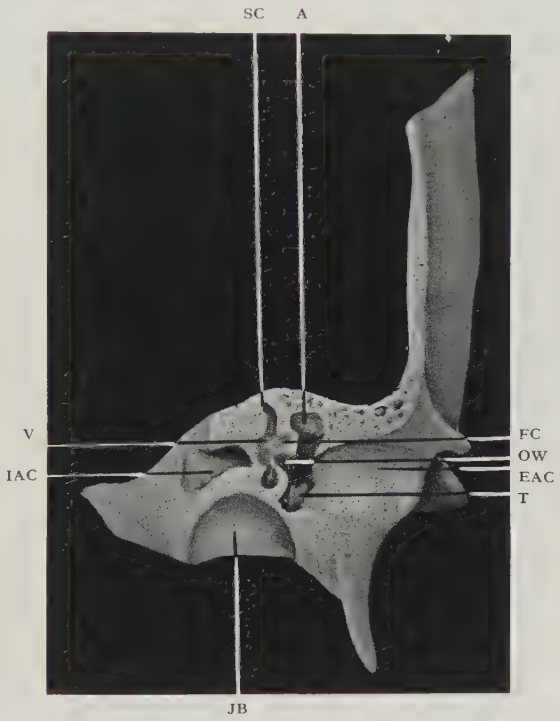

FIG. 47. RIGHT TEMPORAL BONE. VERTICAL SECTION THROUGH EXTERNAL AND INTERNAL AUDITORY CANALS, PARALLEL TO CANALS, VIEWED FROM BEHIND.

A, attic; EAC, external auditory canal; FC, Fallopian canal; IAC, internal auditory canal; JB, jugular bulb; OW, oval window; SC, superior semicircular canal; T, tympanic cavity; V, vestibule.

is a dehiscence in the floor, it is very easy to injure the bulb in the course of a radical mastoid operation. The bulb can also be wounded in the performance of a paracentesis of the drum-membrane.

Körner found a dehiscence in the floor of the middle ear in 30 of 449 skulls studied.

The jugular bulb is in relation, above, with the inner end of the external auditory canal, the middle ear, the lower limb of the posterior semicircular canal, the vestibule, and the outer extremity of the internal auditory canal

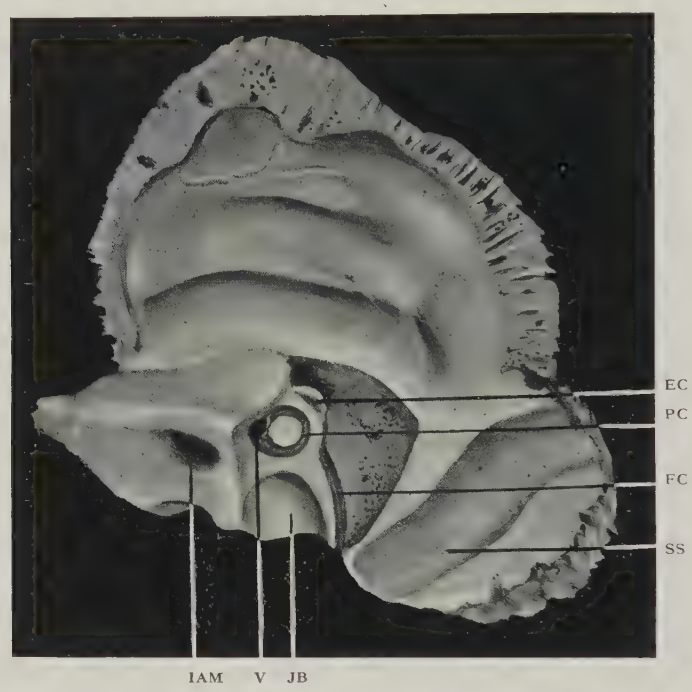

FIG. 48. RIGHT TEMPORAL BONE, VIEWED FROM WITHIN. PORTION OF BASE OF PETROUS PYRAMID REMOVED.

EC, external semicircular canal; FC, Fallopian canal; IAM, internal auditory meatus; JB, jugular bulb; PC, posterior semicircular canal; SS, sigmoid sinus; V, vestibule.

(Figs. 46, 47, 48 and 49). When the dome of the jugular bulb is low, it lies $\frac{1}{8}$ inch below the posterior semicircular canal. But when the dome is high, it may be almost in contact with the canal (Fig. 48). In such cases the bulb may be injured while opening the posterior semicircular canal, or the canal may be injured while opening the bulb.

The bulb is in relation, in front, with the internal carotid artery (Figs. 49 and 50), the aqueductus cochleae, and the structures which pass through the anterior and middle compartments of the jugular foramen, namely the

inferior petrosal sinus, the meningeal branch of the ascending pharyngeal artery, the ninth, tenth and eleventh cranial nerves, and the posterior meningeal artery. Posteriorly, the bulb is in relation to the horizontal limb

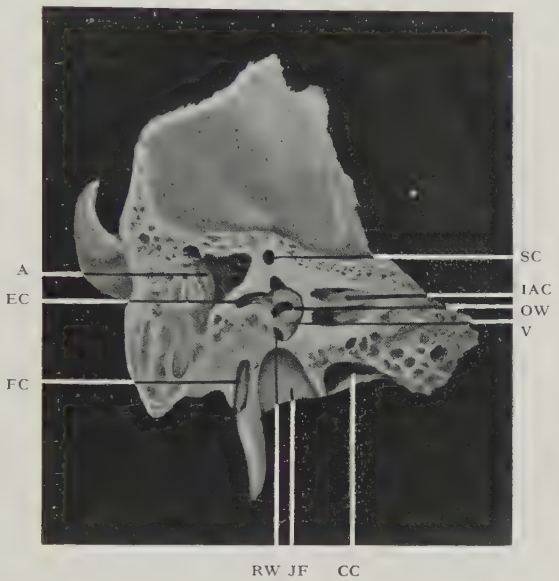

FIG. 49. LEFT TEMPORAL BONE. VERTICAL SECTION FROM TIP OF MASTOID PROCESS TO APEX OF PETROUS PYRAMID, VIEWED FROM BEHIND.

A, aditus; CC, carotid canal; EC, external semicircular canal; FC, Fallopian canal; IAC, internal auditory canal; JF, jugular fossa; OW, oval window; RW, round window; SC, superior semicircular canal; V, vestibule.

of the sigmoid sinus. Internally, it is in relation to the basilar plate of the occipital bone. Externally, it is in relation to the descending or third portion of the facial nerve (Figs. 49, 50, and 51). The facial nerve is only 1 mm. away from the outer wall of the bulb. In operations on the jugular bulb, it is very important to remember the relationship of the facial nerve to the bulb. The bulb is also in relation, externally, to the styloid process (Figs. 49 and 50). It is sometimes necessary to remove the styloid process, in order to expose the bulb from the outer side.

The jugular bulb is continuous with the internal jugular vein. The jugular vein passes out of the skull through the jugular foramen, or posterior

lacerated foramen. The jugular foramen is formed by the jugular notches of the petrous and occipital bones. The jugular foramen is divided by two fibrous tissue septa into three compartments, an antero-internal, a middle,

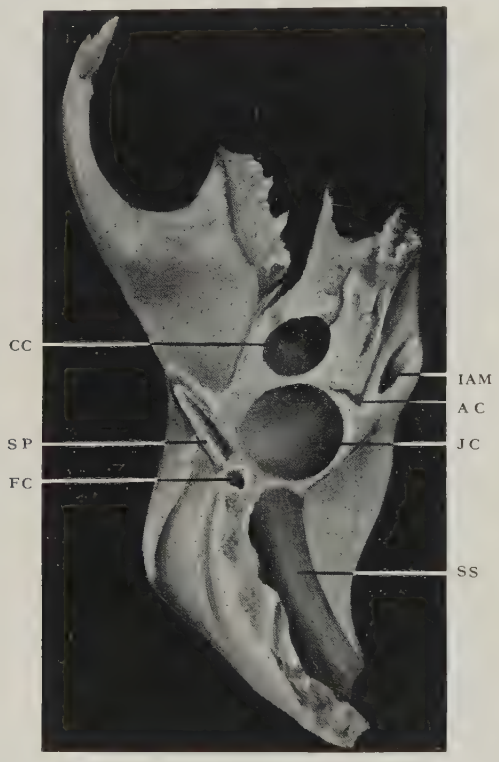

FIG. 50.

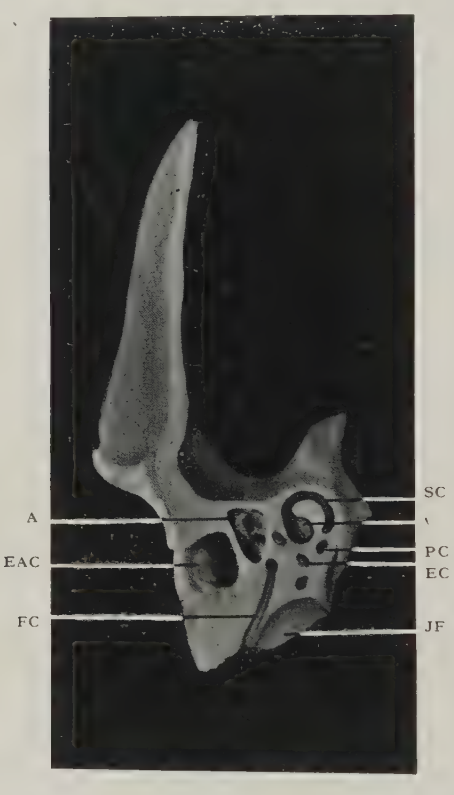

FIG. 51.

FIG. 50. RIGHT TEMPORAL BONE, VIEWED FROM BELOW.

AC, aqueductus cochleae; CC, carotid canal, FC, stylomastoid foramen; IAM, internal auditory meatus; JB, jugular bulb; SP, styloid process; SS, sigmoid sinus.

FIG. 51. LEFT TEMPORAL BONE. VERTICAL SECTION THROUGH EXTERNAL AUDITORY CANAL AT RIGHT ANGLES TO POSTEROSUPERIOR MARGIN OF PETROUS PYRAMID, VIEWED FROM BEHIND.

A, aditus; EAC, external auditory canal; EC, external semicircular canal; FC, Fallopian canal; JF, jugular fossa; PC, posterior semicircular canal; SC, superior semicircular canal; V, vestibule.

and a posteroexternal compartment. The antero-internal compartment transmits the inferior petrosal sinus and a meningeal branch of the ascending pharyngeal artery. The middle compartment transmits the ninth, tenth

and eleventh cranial nerves. The posteroexternal compartment transmits the internal jugular vein (Fig. 52).

According to Braislin, the jugular bulb is much shallower in an infant

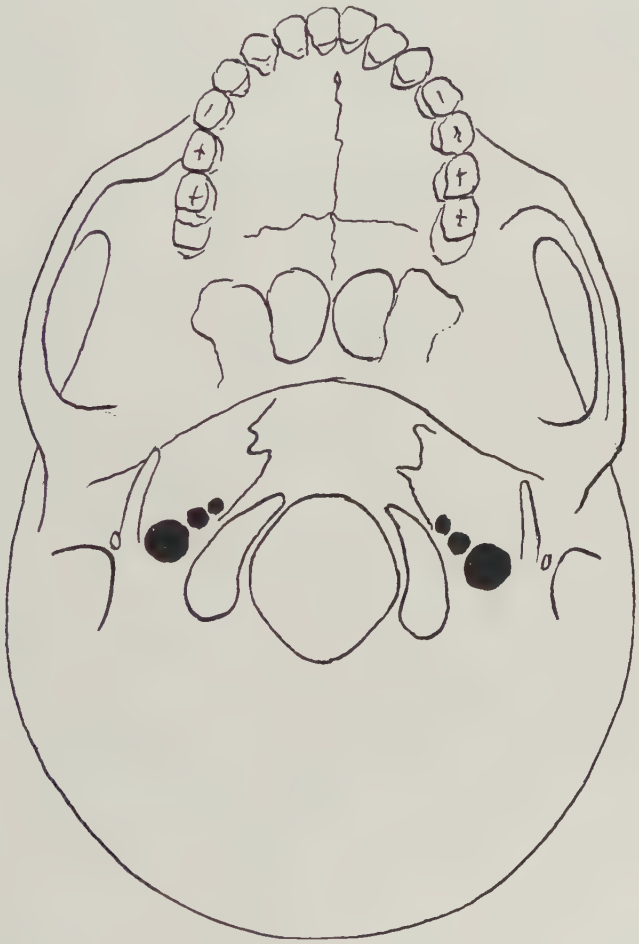

FIG. 52. ARRANGEMENT OF THREE COMPARTMENTS OF JUGULAR FORAMEN.
1. Anterointernal compartment transmits inferior petrosal sinus. 2. Middle compartment transmits ninth, tenth and eleventh cranial nerves. 3. Posteroexternal compartment transmits internal jugular vein.

than in an adult. The jugular foramen in the infant is situated about midway between the rim of the foramen magnum and the periphery of the cerebellar fossa. In the adult, the jugular foramen is more medially situated, and its

course through the bone is directed more outward. The facial nerve overlies the bulb in the infant just as in the adult. In the adult the bony floor of the external auditory canal is external to the bulb. In the infant, the bony

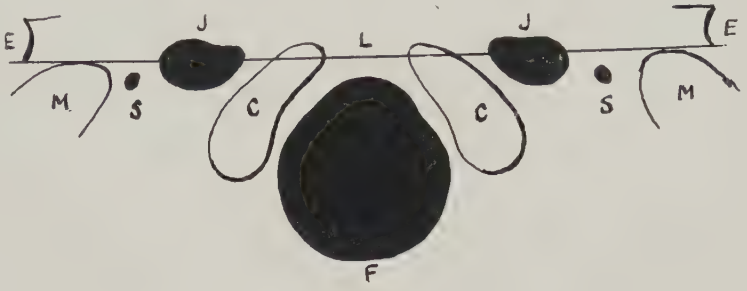

FIG. 53. LINE JOINING ANTERIOR MARGINS OF BOTH MASTOIDS (L). FORAMEN MAGNUM AND CONDYLOID PROCESSES ARE BEHIND THIS LINE. LINE DIVIDES JUGULAR FORAMEN. (*After Feiermann.*)

C, condyle; E, external auditory canal; F, foramen magnum; J, jugular foramen; M, mastoid process; S, stylomastoid foramen.

floor of the canal is wanting and the bulb is much easier of access than in the adult.

In children up to the age of five years, the bulb is more accessible to infection from the middle ear than in the adult, on account of the lesser density of its bony covering.

The relations between the jugular fossa and the following points on the base of the skull vary in different heads: 1, the bony external auditory canal; 2, the anterior margin of the mastoid process; 3, the stylomastoid foramen; 4, the foramen magnum; 5, the occipital condyles.

The line which joins the anterior margins of the two mastoid processes is fairly constant. The stylomastoid foramen usually lies on this line. The exit of the facial nerve is in the plane of the anterior margin of the mastoid. It is about 1 cm. above the tip of the mastoid, at about the same depth as the upper edge of the digastric muscle. The posterior edge of the external auditory canal also coincides with this line.

In some heads, the entire foramen magnum and the condyloid processes are behind this line (Fig. 53). Sometimes the line passes through the anterior third of the foramen magnum (Fig. 54). The line may divide the jugular

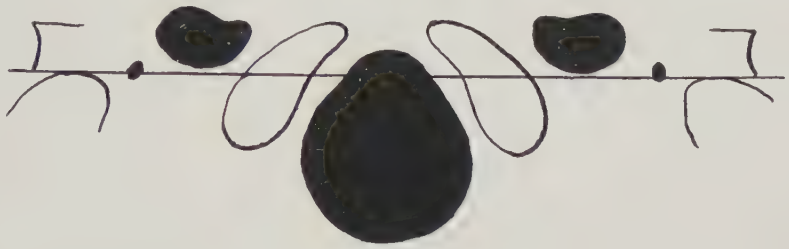

FIG. 54. LINE THROUGH ANTERIOR BORDER OF BOTH MASTOIDS PASSES THROUGH ANTERIOR PART OF FORAMEN MAGNUM, AND DIVIDES CONDYLES IN HALF. TOUCHES POSTERIOR MARGIN OF JUGULAR FORAMEN. (*After Feiermann.*)

foramen in half. It may lie at the posterior margin of the jugular foramen, or it may lie at an appreciable distance behind the jugular foramen (Fig. 55).

The facial nerve is always external to the jugular fossa and somewhat behind it.

When the line joining the anterior margins of the two mastoids is behind the jugular fossa, the conditions are most favorable for doing a

FIG. 55. LINE THROUGH ANTERIOR MARGINS OF BOTH MASTOIDS LIES AT A CONSIDERABLE DISTANCE BEHIND JUGULAR FORAMEN. (*After Feiermann.*)

Pifft operation, as the jugular fossa is opposite the external auditory canal, and in front of the facial nerve. In the Pifft operation, the jugular bulb is opened by removing the floor of the external auditory canal and the floor of the middle ear.

From an examination of a large number of skulls, Feiermann found that the measurements at the base of the skull bear no relationship to the form of the top of the skull. But he found a direct relationship between the measurements at the base of the skull and the width of the skull between the outer surfaces of both mastoid processes.

Skulls in which the width between the outer surfaces of both mastoids was between 105 and 108 mm. had a measurement of 12 to 13 mm. from the outer margin of the bony external auditory canal to the outer margin of the jugular fossa.

Skulls in which the width between the outer surfaces of both mastoids was between 137 and 140 mm., had a measurement of 16 to 17 mm. from the outer margin of the bony external auditory canal to the outer margin of the jugular fossa.

The greatest measurement was 23 mm. and the smallest was 8 mm. This measurement is of value in the performance of the Piffl operation.

In the Grunert operation, the measurement from the outer surface of the mastoid to the outer margin of the condyloid process is important.

In skulls with a width of 105 to 108 mm. between the outer surfaces of the mastoids, the distance between the outer surface of the mastoid and the outer margin of the condyloid process was 30 to 31 mm.

In skulls with a width of 137 to 140 mm. between the outer surfaces of the mastoids, the distance between the outer surface of the mastoid and the outer margin of the condyloid process was 42 to 43 mm. The smallest measurement was 24 mm. and the greatest was 47 mm.

The jugular foramina are often unequal on the two sides. Zuckerkandl described a skull in which the right jugular foramen was eight times as wide as the left one. The left jugular foramen was only 3 mm. wide.

Linser found, in an examination of 1022 skulls, that the average size of the venous outlet in the jugular foramen was 7 to 9 mm. In 29 skulls the

venous opening was 3 to 4 mm. in diameter, the opposite one being three or four times as large.

The largest of the vessels which empty into the jugular bulb, with the exception of the sigmoid sinus, is the inferior petrosal sinus. This opens into the anterior wall of the bulb. Sometimes the vein of the aqueductus cochleae empties into the bulb. Occasionally there is a vein from the occipital sinus to the bulb. Near the entrance of the inferior petrosal sinus, there are sometimes seen several small veins passing from the jugular bulb, between the ninth, tenth and eleventh nerves, to the descending veins in the neighborhood. There may be a communication between the jugular bulb and the anterior condyloid vein, when the posterior condyloid vein is missing.

The Posterior Occipital Sinus. The posterior occipital sinus lies against the inner surface of the occipital bone. It extends in a sagittal direction from the internal occipital protuberance to the posterior margin of the foramen magnum. It runs along the line of attachment of the falx cerebelli to the occipital bone. It begins above at the torcular Herophili, and when it reaches the posterior margin of the foramen magnum, it splits into two branches, called the marginal sinuses. Each marginal sinus runs along the corresponding lateral margin of the foramen magnum, and empties into the lower portion of the sigmoid sinus, just above the entrance of the latter into the jugular bulb. The occipital sinus averages 2 mm. in diameter.

The marginal sinuses are occasionally wanting. When this occurs the posterior occipital sinus ends in the plexus circularis of the foramen magnum, and through it, communicates with the posterior longitudinal spinal plexus. According to Streit this occurs in 25 per cent of the cases.

Sometimes there are two posterior occipital sinuses. It is missing altogether in 20 per cent of the cases.

Instead of beginning at the torcular, the occipital sinus may begin in one of the lateral sinuses (Fig. 56). It may split into two branches high up,

or there may be two separate occipital sinuses, in which case each may take its origin in one of the lateral sinuses (Fig. 57).

The occipital sinus may be very large, and the lateral sinus of one side

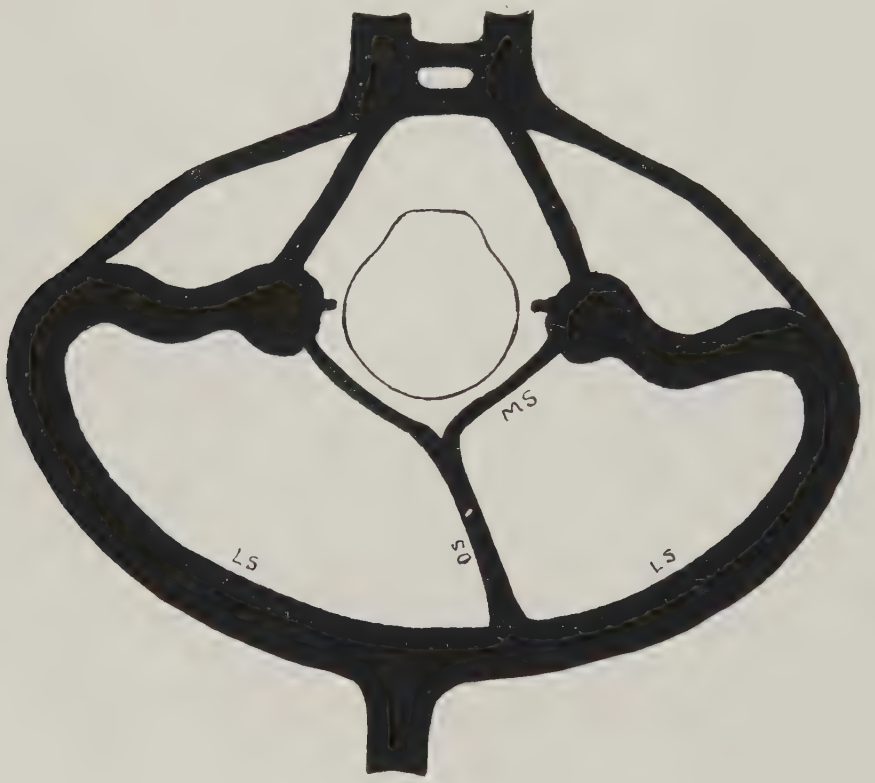

FIG. 56. OCCIPITAL SINUS JOINS LEFT LATERAL SINUS.
LS, lateral sinus; MS, marginal sinus; OS, occipital sinus.

may be very small, the function of the latter being taken up, in part, by the former. When this occurs, the blood stream passes from the superior longitudinal sinus through the enlarged occipital sinus to the jugular bulb (Fig. 58).

The venous connections of the posterior occipital sinus are the following:

1. Veins from the under surface of the cerebellum, from the pons and from the medulla, empty into the occipital sinus.

2. Venous connections between the occipital sinus and the jugular bulb.

3. The occipital sinus is connected with the venous ring which surrounds the foramen magnum (Fig. 59). The latter communicates on one side with the deep cervical veins, and on the other side with the veins of the spinal

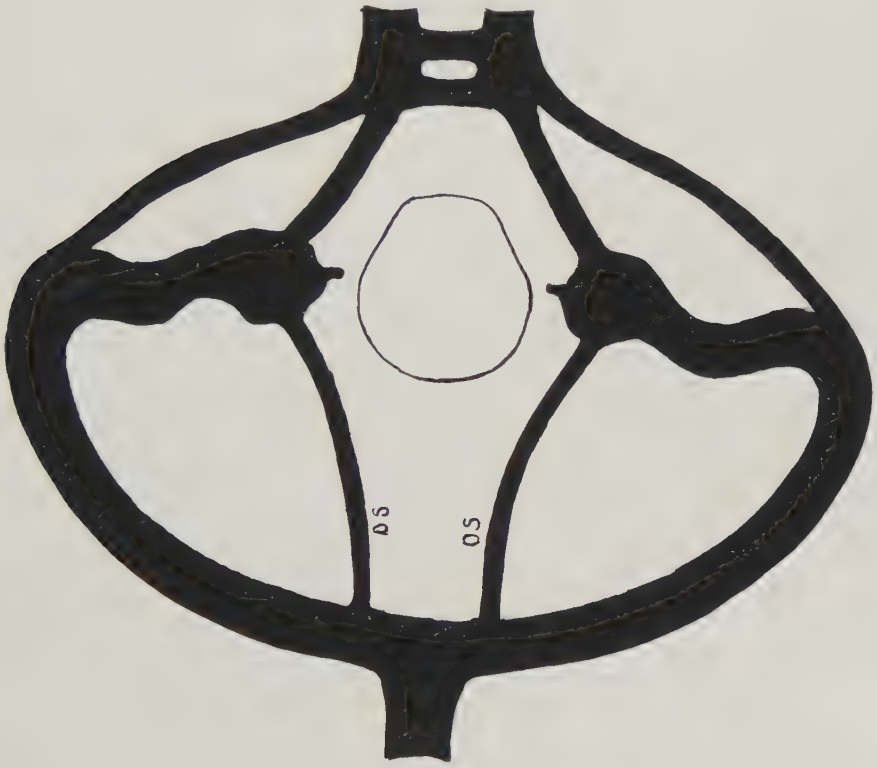

FIG. 57. TWO OCCIPITAL SINUSES, EACH EMPTYING INTO LATERAL SINUS OF ITS OWN SIDE.
OS, occipital sinus.

canal. The deep cervical veins join the vertebral vein, and the latter empties into the innominate vein. In this way, the posterior occipital sinus empties into the innominate vein.

Streit examined a large number of skulls, and found that he could divide them into five types, depending on the pathway through which the

blood passed from the superior longitudinal sinus to the internal jugular veins (Fig. 60). They are:

Type 1. The blood column divides at the posterior extremity of the

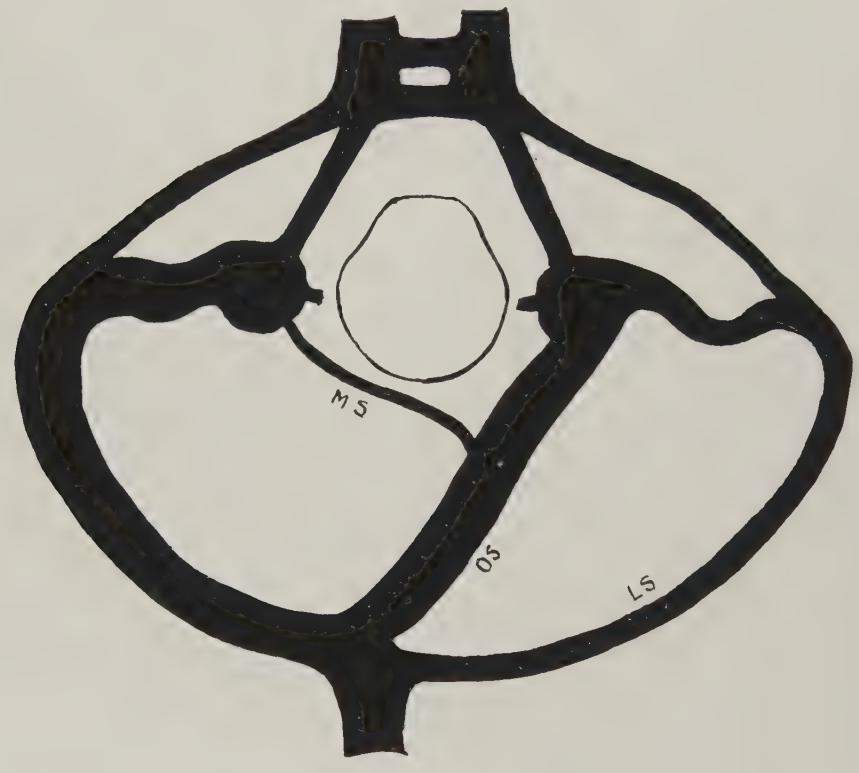

FIG. 58. LEFT LATERAL SINUS VERY SMALL. OCCIPITAL SINUS VERY LARGE AND PASSES FROM TORCULAR TO LEFT JUGULAR BULB.

LS, left lateral sinus; MS, marginal sinus; OS, occipital sinus.

superior longitudinal sinus, and flows through both lateral sinuses and the occipital sinus, to the internal jugular veins, the greater column of blood passing through the lateral sinuses.

Type 2. The blood column divides at the posterior extremity of the superior longitudinal sinus, and passes on one side, through the lateral and occipital sinus, and on the other side, through the lateral or occipital sinus, to the internal jugular vein.

Type 3. The blood column divides at the posterior extremity of the superior longitudinal sinus, and passes on one side through the occipital sinus, and on the other side, through the lateral sinus to the internal jugular veins.

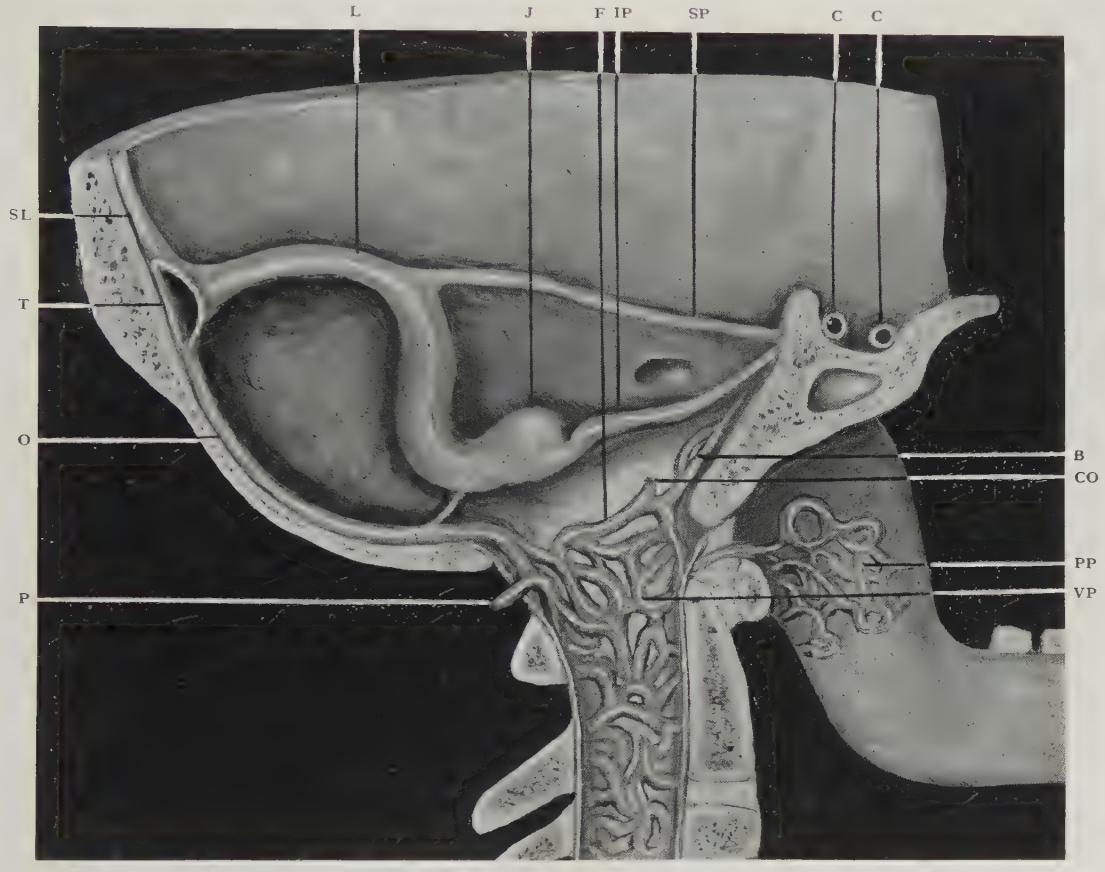

FIG. 59. COMMUNICATIONS OF VERTEBRAL PLEXUS.

B, basilar plexus; C, circular sinus; CO, communication between vertebral plexus and jugular bulb, through anterior condyloid foramen; F, plexus of the foramen magnum; IP, inferior petrosal sinus; J, jugular bulb; L, lateral sinus; O, occipital sinus; P, communication between vertebral plexus and deep neckveins; PP, pterygoid plexus; SL, superior longitudinal sinus; SP, superior petrosal sinus; T, torcular; VP, vertebral plexus.

Type 4. The blood column divides at the posterior extremity of the superior longitudinal sinus, and passes through both lateral sinuses to the internal jugular veins. The occipital sinus is wanting.

Type 5. The blood column flows entirely, or almost entirely, to one side, through the lateral and occipital sinus, or through the lateral sinus alone, to the internal jugular. In this type, the opposite jugular foramen is very small.

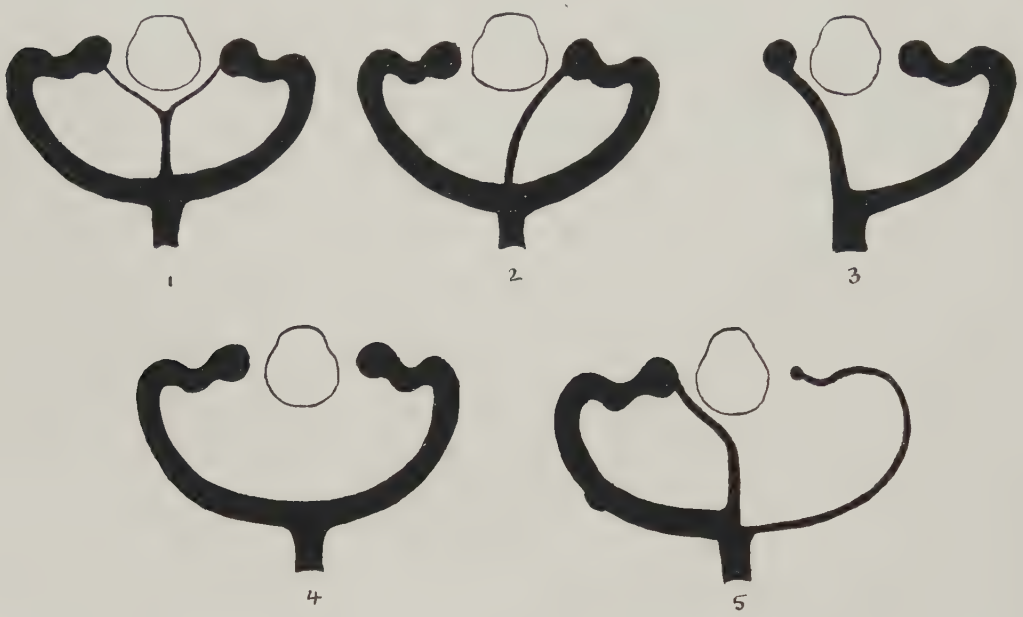

FIG. 60. FIVE TYPES OF CIRCULATION FROM SUPERIOR LONGITUDINAL SINUS TO INTERNAL JUGULAR VEIN, DESCRIBED BY STREIT.

1. From superior longitudinal sinus through both lateral sinuses and occipital sinus to internal jugular vein, greater column of blood passing through lateral sinuses.

2. From superior longitudinal sinus through lateral and occipital sinus on one side, and through lateral or occipital sinus on other side.

3. From superior longitudinal sinus through lateral sinus on one side, and through occipital sinus on other side.

4. From superior longitudinal sinus through both lateral sinuses. Occipital sinus wanting.

5. From superior longitudinal sinus, entirely, or almost entirely, through lateral and occipital sinus on one side. Opposite lateral sinus rudimentary.

THE SUPERIOR PETROSAL SINUS. The superior petrosal sinus carries blood from the cavernous sinus to the lateral sinus. It begins at the posterior extremity of the cavernous sinus and passing backward, outward, and slightly upward, ends in the knee of the lateral sinus. It is lodged in a groove on the posterosuperior margin of the petrous pyramid, and lies along the

line of attachment of the tentorium cerebelli to the petrous pyramid. It is at the line of separation of the middle from the posterior fossa of the skull.

The Gasserian ganglion lies above the anterior extremity of the superior petrosal sinus.

Absence of the superior petrosal sinus on one side was found by Knott in 3 skulls. Hyrtl found one superior petrosal sinus absent in 1 skull.

A dehiscence in the superior petrosal sulcus leading into the mastoid cells, at the point where the superior petrosal sulcus joins the sigmoid sulcus, was found by Hyrtl in 3 skulls.

The following veins empty into the superior petrosal sinus:

1. The vein of the aqueductus vestibuli empties into the superior petrosal sinus.

2. Veins from the middle ear pass through the petrosquamous suture and enter the superior petrosal sinus.

3. A vein from the diploetic tissue about the semicircular canals sometimes leads to the superior petrosal sinus.

4. There are venous connections between the superior petrosal sinus and the middle meningeal vein. Zuckerkandl describes a groove, which he found to be fairly constant, passing from the foramen spinosum to the upper margin of the petrous pyramid. This groove contains a vein connecting the superior petrosal sinus with the main trunk of the middle meningeal vein.

5. Sometimes a subarcuate vein comes out of the subarcuate fossa and enters the superior petrosal sinus.

6. There may be anastomoses between the superior and inferior petrosal sinuses in front of the internal auditory meatus.

7. The great anastomotic vein of Trolard sometimes empties into the superior petrosal sinus. This vein runs in the posterior cleft of the fissure of Sylvius. It arises from one of the tributaries of the middle cerebral vein,

and collects the blood from the surface of the parietal lobes. It is continuous with a superior cerebral vein which ascends to the superior longitudinal sinus. In this way there is formed an anastomosis between the sinuses in the vault of the skull and the sinuses at the base of the skull (Fig. 17). Sometimes the vein of Trolard empties into the sigmoid sinus.

The Inferior Petrosal Sinus. The inferior petrosal sinus passes from the posterior extremity of the cavernous sinus to the jugular bulb or the internal jugular vein. It lies in the petro-occipital suture, which is the groove formed by the junction of the lower margin of the petrous pyramid with the basilar process of the occipital bone. It passes through the antero-internal compartment of the jugular foramen, and ends either in the anterior surface of the jugular bulb, or in the upper end of the internal jugular vein.

In 9 skulls examined by Grunert, he found the inferior petrosal sinus emptying into the lower part of the bulb 3 times, into the central part of the bulb twice, and into the jugular vein 4 times. It never ends in the upper part of the bulb.

As it passes through the jugular foramen, it is accompanied by a meningeal branch of the ascending pharyngeal artery. It is separated by a fibrous septum from the middle compartment of the jugular foramen, which contains the ninth, tenth and eleventh nerves.

The inferior petrosal sinus is semicircular on cross section. It is shorter and wider than the superior petrosal sinus, and at its point of entrance into the bulb, it is provided with a valve, according to Ballance.

The superior and inferior petrosal sinuses are in contact with each other at their anterior extremities, but diverge posteriorly. The superior petrosal sinus inclines slightly upward, and the inferior petrosal sinus inclines markedly downward.

The inferior petrosal sinus is in relation above with the seventh and eighth nerves. Internally, it is in relation with the lower surface of the lateral cerebellar hemisphere.

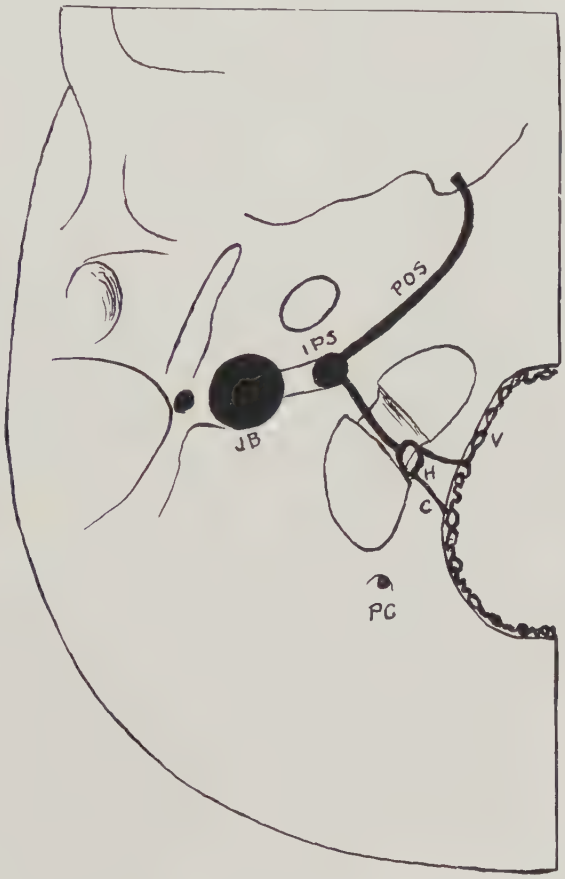

FIG. 61. VENOUS ANASTOMOSES OF INFERIOR PETROSAL SINUS. (*After Charpy.*)

C, condyloid sinus in anterior condyloid canal; H, venous ring around hypoglossal nerve; IPS, inferior petrosal sinus; JB, jugular bulb; PC, posterior condyloid foramen; POS, petrooccipital sinus, which forms anastomosis between inferior petrosal sinus and carotid sinus; V, venous plexus of foramen magnum.

The inferior petrosal sinus and the posterior end of the cavernous sinus may be infected in apical necrosis of the petrous pyramid.

The following veins empty into the inferior petrosal sinus: (1) the vein of the aqueductus cochleae; (2) the vein of the subarcuate fossa; (3) the vein of the aqueductus vestibuli, and (4) the internal auditory vein.

There is a communication between the inferior petrosal sinus and a venous plexus from the cavernous sinus which lies on the clivus. This plexus is called the anterior occipital plexus, or basilar plexus. There is a communication between the inferior petrosal sinus and the carotid plexus by means of the petro-occipital sinus. This sinus is lodged in the petro-occipital suture, below the inferior petrosal sinus. There is a communication between the inferior petrosal sinus and the vertebral plexus by means of a vein which passes through the anterior condyloid canal. According to Henle, there are two veins in the anterior condyloid canal, which pass from the vertebral plexus to the inferior petrosal sinus. At the entrance of the anterior condyloid canal, there is a venous plexus called the circellus venosus hypoglossi, from which blood is carried downward through the pharyngeal vein. It is connected to the venous plexus about the foramen magnum (Fig. 61).

The Cavernous Sinus. The cavernous sinuses are situated in the middle fossa of the skull and lie on each side of the body of the sphenoid bone. They extend from the sphenoidal fissure, in front, to the apex of the petrous pyramid, behind. They are situated between two layers of the dura, and are lined by endothelium. They differ in structure from the other venous sinuses of the skull, in the fact that they consist of a reticulated or erectile structure, very much like the corpus cavernosum of the penis. From this fact, they have received their name. The lumen of the cavernous sinus is narrowed not only by the trabeculae which it contains, but also by the presence within its lumen of a number of nervous and vascular structures, namely the third, fourth, the first and second divisions of the fifth, and the sixth nerves, and the internal carotid artery. Although these structures lie within the lumen of the sinus, they are separated from the blood stream by a layer of endothelium (Fig. 62).

The cavernous sinuses drain the blood anteriorly from the orbits, through the ophthalmic veins, and anterosuperiorly from the brain, through

the sphenoparietal sinus and the middle cerebral vein, and sometimes the inferior cerebral vein. They empty into the petrosal sinuses, and through these, into the internal jugular veins. The cavernous sinuses are larger

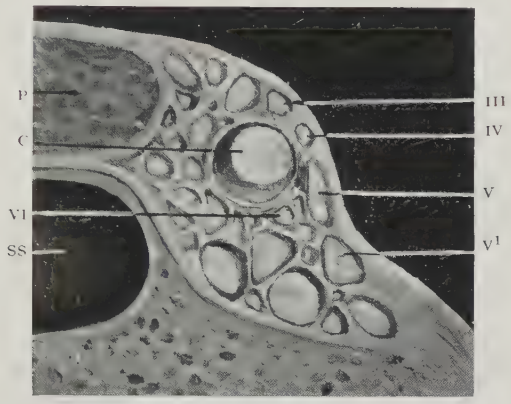

FIG. 62. FRONTAL SECTION OF RIGHT CAVERNOUS SINUS.

C, internal carotid artery; P, pituitary body; SS, sphenoidal sinus; III, third nerve; IV, fourth nerve; V, first division of fifth nerve; V^1, second division of fifth nerve; VI, sixth nerve.

behind than in front. They are 2 to 2¼ cm. long, and about 1 cm. wide. They are roughly quadrilateral on cross section.

In the child, the cavernous sinus consists of a plexus of veins in the dura. By enlargement and fusion of its channels, it is converted into the characteristic sinus of the adult. In old age, some of the trabeculae become absorbed, and the sinus is more simple in its structure.

The reticulated structure of the sinus interferes very markedly with its operative treatment. Simple incision of the sinus is not effective, for if some of the compartments remain unopened, they will continue to spread the infection.

On the inner wall, within the sinus, lies the internal carotid artery, accompanied by filaments of the carotid plexus, and by the sixth nerve. The carotid artery is partly surrounded by the carotid sinus. In the outer wall of

the sinus are the third, fourth, and the first and second divisions of the fifth, nerves.

In the posterior four-fifths of the sinus, the structures from above down-

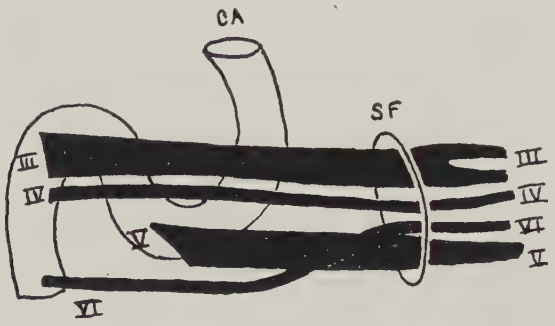

FIG. 63. ARRANGEMENT OF NERVES IN CAVERNOUS SINUS AND IN SPHENOIDAL FISSURE. RIGHT SIDE. SIXTH NERVE BELOW FIRST DIVISION OF FIFTH NERVE IN POSTERIOR PART OF CAVERNOUS SINUS. IT PASSES MEDIAL TO FIRST DIVISION OF FIFTH NERVE IN ANTERIOR PART OF CAVERNOUS SINUS, AND LIES ABOVE FIRST DIVISION OF FIFTH NERVE IN SPHENOIDAL FISSURE. SECOND DIVISION OF FIFTH NERVE NOT INDICATED IN DIAGRAM.

CA, internal carotid artery; SF, sphenoidal fissure; III, third nerve; IV, fourth nerve; V, first division of fifth nerve; VI, sixth nerve.

ward are the third, fourth and first division of the fifth nerve resting against the inner surface of the outer wall. The carotid artery, surrounded by the carotid plexus, and the sixth nerve rest against the inner wall. The cavernous plexus of nerves lies internal to the carotid artery. In the anterior fifth of the sinus, the third, and the first division of the fifth, nerves divide into their branches.

According to Sluder, the sixth nerve, in the sphenoidal fissure, is medial to and above the first division of the fifth nerve. In the cavernous sinus, the sixth nerve is medial to and below the level of the first division of the fifth nerve (Fig. 63).

The outer surface of the cavernous sinus is in relation, in its upper three-quarters, with the inner side of the anterior extremity of the temporosphenoidal lobe. In its lower quarter, it is in relation with the third division of

the fifth nerve. The upper border of the sinus is in relation, in its posterior four-fifths, with the dura covering the third nerve. In its anterior fifth, it is in relation with the lachrymal nerve.

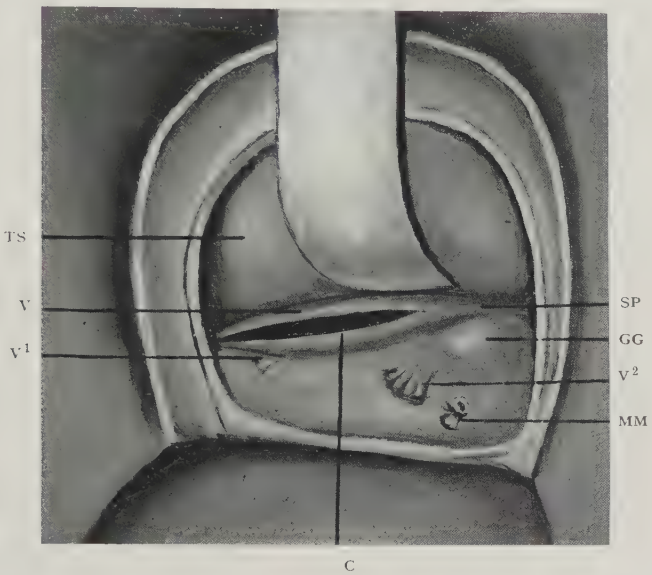

FIG. 64. SURGICAL EXPOSURE OF LEFT CAVERNOUS SINUS. TEMPORAL BONE FLAP REMOVED AS IN HARTLEY-KRAUSE OPERATION. (*After Ballance.*)

C, cavernous sinus opened by a horizontal incision; GG, Gasserian ganglion; MM, middle meningeal artery ligated; SP, superior petrosal sinus; TS, dura mater over temporosphenoidal lobe; V, first division of fifth nerve; V^1, second division of fifth nerve; V^2, third division of fifth nerve.

The inferior border of the sinus is in relation with the third division of the fifth nerve (Fig. 64).

The cavernous sinus overlaps the roof of the sphenoidal sinus. The roof of the sphenoidal sinus is often very thin, and disease of the sphenoidal sinus can easily extend into the cavernous sinus. The bony carotid canal is often seen bulging into the cavity of the sphenoidal sinus, near the postero-external angle of the latter.

a. Circular Sinus. The two cavernous sinuses communicate with each other by means of the anterior and posterior intercavernous sinuses. These

two sinuses form the circular sinus. They lie in the sella turcica, and surround the hypophysis. They receive branches from the hypophysis and from the dura. The circular sinus allows a rapid spread of infection from one cavernous sinus to the other.

The part of the internal carotid artery which lies within the cavernous sinus is the third or terminal portion. After the artery leaves the petrous pyramid, it curves upward to enter the cavernous sinus. Here it bends forward, following the carotid groove along the lateral surface of the body of the sphenoid, from the posterior clinoid process to the anterior clinoid process, where it again curves upward and slightly backward to perforate the dura which forms the roof of the cavernous sinus. After piercing the dura, it passes between the optic and oculomotor nerves to the internal extremity of the lateral cerebral fissure, where it divides into its terminal branches. The carotid plexus of veins surrounds the artery as far as the lower end of the carotid canal, where it empties into the internal jugular vein.

There are two plexuses of nerves accompanying the internal carotid artery, the carotid plexus and the cavernous plexus. The carotid plexus is situated on the outer side of the carotid artery just as the artery enters the cavernous sinus. The cavernous plexus lies below and internal to the artery, within the cavernous sinus.

The cavernous sinus is formed, in front, by the junction of the superior and inferior ophthalmic veins and the sphenoparietal sinus.

b. The Sphenoparietal Sinus. The sphenoparietal sinus, or sinus of Breschet (Fig. 16), is composed of two branches, a vertical descending branch which passes from the superior longitudinal sinus to the external angle of the lesser wing of the sphenoid bone, in the neighborhood of the frontoparietal suture, and a horizontal branch, which is hidden under the lesser wing of the sphenoid. It ends in the anterior end of the cavernous

sinus. It is about 3 mm. in diameter. It receives tributaries from the diploe, from the dura, and from the brain.

c. Ophthalmic Veins. There are two ophthalmic veins, superior and inferior. The superior ophthalmic vein is the larger of the two. It commences near the root of the nose as a continuation of the frontal vein, where it communicates with the angular vein. It lies under the roof of the orbit, crossing the optic nerve from within outward, and gains the inner end of the sphenoidal fissure, where it empties into the cavernous sinus. The inferior ophthalmic vein lies near the floor of the orbit, and enters the cavernous sinus either separately or in common with the superior ophthalmic vein. The inferior ophthalmic vein sends a communicating branch down through the sphenomaxillary fissure to the pterygoid plexus. Sometimes this branch to the pterygoid plexus is the principal termination of the inferior ophthalmic vein.

The communications between the cavernous sinuses and the neighboring veins are very numerous and complex. This multiplicity of communications allows of spread of infection to the cavernous sinus from various parts of the head (Fig. 65):

1. The cavernous sinus communicates with the facial vein through the superior ophthalmic and angular veins.

2. The cavernous sinus communicates with the pterygoid plexus through the inferior ophthalmic vein. A branch from the ophthalmic vein passes to the pterygoid plexus through the sphenomaxillary fissure. The cavernous sinus also communicates with the pterygoid plexus through several vessels which pass through the foramen ovale, the foramen spinosum, and the middle lacerated foramen. The pterygoid plexus, in turn, empties into the external and internal jugular veins.

3. The cavernous sinus communicates with the superior longitudinal sinus through the sphenoparietal sinus of Breschet.

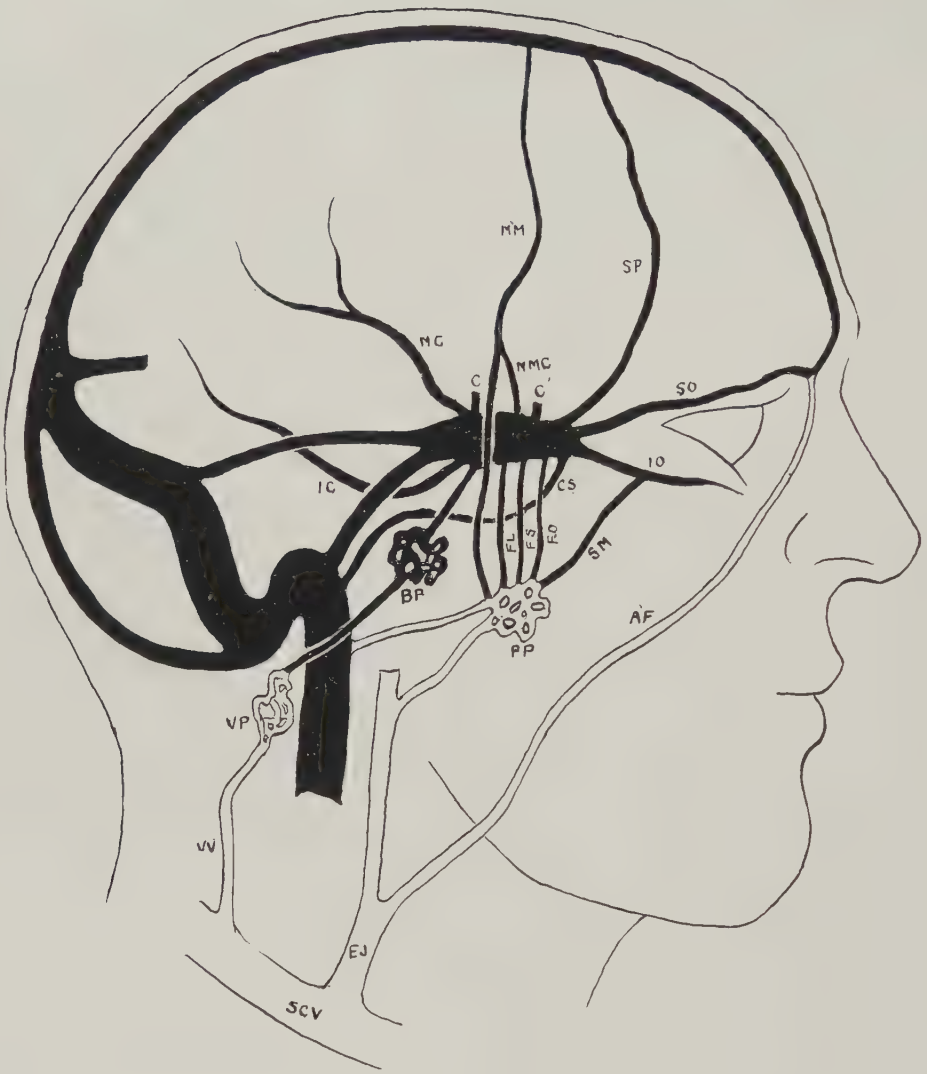

FIG. 65. COMMUNICATIONS OF CAVERNOUS SINUS.

AF, anterior facial vein; BP, basilar plexus; C, circular sinus; CS, carotid sinus; EJ, external jugular vein; FL, communicating vessel passing through foramen lacerum; FO, communicating vessel passing through foramen ovale; FS, communicating vessel passing through foramen spinosum; IC, inferior cerebral vein; IO, infraorbital vein; MC, middle cerebral vein; MM, middle meningeal vein; MMC, communication between middle meningeal vein and cavernous sinus; PP, pterygoid plexus; SCV, subclavian vein; SM, communication between infraorbital vein and pterygoid plexus; SO, supraorbital vein; SP, sphenoparietal sinus; VP, vertebral plexus; VV, vertebral vein.

4. The cavernous sinus communicates with the middle meningeal vein, and the middle and inferior cerebral veins.

5. The cavernous sinus communicates with the lateral sinus through the superior petrosal, and with the jugular bulb through the inferior petrosal.

6. The cavernous sinus communicates, through the basilar plexus (a venous plexus which lies on the clivus), with the vertebral plexus. From here, the blood reaches the deep cervical veins.

7. The cavernous sinus communicates with the carotid sinus, which accompanies the internal carotid artery. The carotid sinus receives branches from the middle ear, and empties into the internal jugular vein.

8. The two cavernous sinuses communicate with each other through the circular sinus.

d. The Pterygoid Plexus of Veins. The pterygoid plexus of veins plays so important a rôle in the spread of infections to the venous sinuses at the base of the skull, that it deserves a detailed description.

It lies in the zygomatic fossa, and accompanies the internal maxillary artery. It consists of a network of large veins covering both surfaces of the external pterygoid muscle, and the inner surface of the internal pterygoid muscle (Fig. 66). It receives several deep temporal veins from the temporal muscle, the middle meningeal veins, the sphenopalatine vein, and communicating branches from the inferior ophthalmic vein and cavernous sinus. The sphenopalatine vein carries most of the blood from the nasal mucosa to the pterygoid plexus. It receives small communicating vessels from the cavernous sinus, which pass through the foramen ovale, foramen spinosum and middle lacerated foramen.

The pterygoid plexus sends branches to the anterior and posterior facial veins. Through these vessels it reaches the internal and external jugular veins. It also reaches the internal jugular vein through the pharyngeal vein.

The Carotid Sinus. The carotid sinus is the name given to the plexus of veins which surrounds the internal carotid artery, as the latter passes through its canal in the petrous pyramid. It is a venous plexus rather

FIG. 66. PTERYGOID PLEXUS OF VEINS.

A, angular vein; AF, anterior facial; C, communicating veins between cavernous sinus and pterygoid plexus; F, common facial; DAF, deep anterior facial; DPF, deep posterior facial; DT, deep temporal; EJ, external jugular; F, frontal vein; IJ, internal jugular; IO, inferior ophthalmic, L, labial vein; MM, middle meningeal; N, nasal vein; P, pharyngeal; PF, posterior facial; PP, pterygoid plexus; PSF, posterior superficial facial; SAF, superficial anterior facial; SO, superior ophthalmic; SP, sphenopalatine; ST, superficial temporal.

than a sinus. In older individuals, the venous spaces fuse to form larger compartments similar to the cavernous sinus (Fig. 67). Before the artery enters its canal, the plexus is situated in the arterial adventitia. The network

FIG. 67. INTERNAL CAROTID ARTERY SURROUNDED BY CAROTID VENOUS PLEXUS. (*After Haike.*)

is thickest at the lower entrance of the artery into the carotid canal and at its exit from the canal into the cranial cavity. The vertical part of the artery and the knee have only a few venous filaments (Figs. 68 and 69).

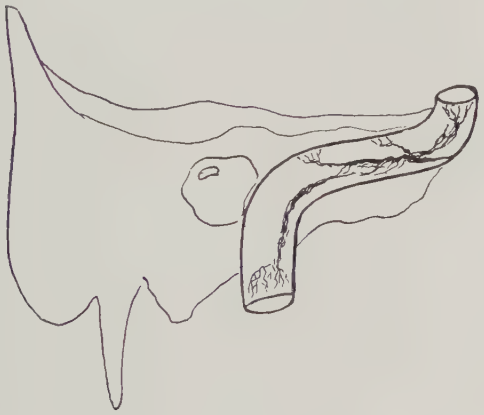

FIG. 68. CAROTID SINUS. VENOUS NETWORK ON LATERAL WALL OF INTERNAL CAROTID ARTERY. (*After Haike.*)

FIG. 69. VENOUS NETWORK ON MEDIAL WALL OF INTERNAL CAROTID ARTERY. (*After Haike.*)

The carotid sinus is connected to the middle ear vessels by the caroticotympanic veins. It sometimes receives vessels from the bone. It communi-

cates with the internal jugular vein on one side, and with the cavernous sinus on the other.

It constitutes a preformed pathway from the middle ear to the interior of the skull. In caries of the walls of the middle ear, there is a wide communication between the middle ear and the carotid sinus.

The Anterior Occipital Sinus. The anterior occipital sinus is also known as the basilar plexus. It lies on the basilar process of the occipital bone, and communicates with both inferior petrosal sinuses and the uppermost of the anterior spinal plexuses. The latter is called the plexus of the foramen magnum (Figs. 16 and 59).

The Petrosquamous Sinus. Luschka gives the following description of the petrosquamous sinus:

"The lateral sinus runs in earliest fetal life from the internal occipital protuberance across the upper surface of the petrous pyramid and passes out of the skull through the foramen jugulare spurium. As this foramen becomes gradually obliterated, and as the branch of the primitive jugular vein which is to become the future internal jugular vein develops, that portion of the lateral sinus is formed, which corresponds to the fossa sigmoidea of the mastoid portion of the temporal bone. The original course of the lateral sinus does not always disappear entirely, but some traces of it remain all through life. It manifests itself in the temporal bone in the form of a groove along the petrosquamous suture."

The petrosquamous sinus is a remnant of the primitive head vein. It begins behind, at the lateral sinus between the torcular and the point of entrance of the superior petrosal sinus, runs along the upper surface of the petrous pyramid, and reaches the exterior of the skull through the spurious jugular foramen, or through the foramen spinosum by way of the middle meningeal vein.

The petrosquamous sulcus is seen in many skulls. Sometimes it is converted into a bone canal in part. According to Krause, the petrosquamous sinus ends in the middle meningeal vein. The sinus may have several bends,

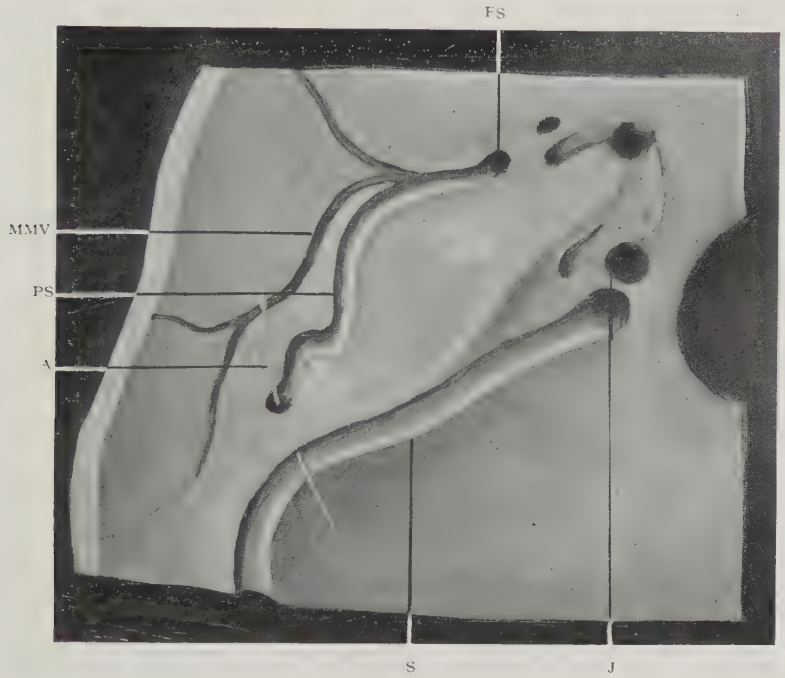

FIG. 70. (*After Streit.*)

A, probe passing from petrosquamous sinus to lateral sinus; FS, foramen spinosum; J, jugular foramen; MMV, middle meningeal vein; PS, petrosquamous sinus; S, sigmoid sinus.

and may lie, in part, over the roof of the middle ear. The size of the sinus varies very much.

Zuckerkandl described a skull in which the petrosquamous sinus was 5.5 mm. wide. It ran from the sigmoid sinus to the foramen spinosum. There were four outlets to the sigmoid sinus in this case:

1. Through the petrosquamous sinus to the middle meningeal vein
2. Through the spurious jugular foramen
3. Through the mastoid foramen

4. Through an anastomosis between the middle meningeal vein and the veins of the surface of the skull.

Cheatle found a persistent spurious jugular foramen in 23 skulls out of

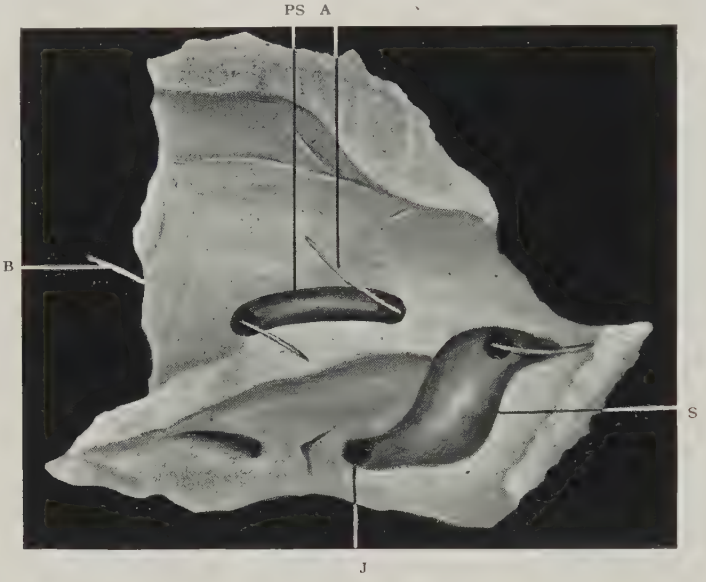

FIG. 71. (*After Streit.*)

A, probe passing from petrosquamous sinus to sigmoid sinus; B, probe passing from petrosquamous sinus through spurious jugular foramen; J, jugular foramen; PS, petrosquamous sinus; S, sigmoid sinus.

2585 which he examined. In these 23 skulls, 3 were in the glenoid fossa, 3 in the zygomatic process, 6 at the base of the malar bone, and 11 just external to the Glaserian fissure.

Streit reported 2 cases of petrosquamous sinus (Figs. 70 and 71).

Blood Pathways from the Sinuses to the Outside of the Skull

Main Blood Pathway. The main pathway of the blood from the intracranial sinuses to the outside of the skull is through the internal jugular veins. But there are many accessory pathways, some of which are in use under normal conditions, and some of which are utilized only when there is an obstruction in some part of the intracranial sinuses.

Accessory Pathways. Some of these accessory pathways are:

1. Through the vertebral veins. The inferior petrosal sinuses, the anterior occipital or basilar plexus, and the posterior occipital sinuses communicate with the vertebral plexus, which lies within the spinal canal. The jugular bulb also communicates with the vertebral plexus through a vein which passes through the anterior condyloid foramen. The vertebral plexus empties the vertebral vein, which, in turn, opens into the beginning of the innominate vein.

2. Through the emissary veins. There are four sets of these, the mastoid emissary, the occipital emissary, the parietal emissary or emissary of Santorini, and the condyloid emissary. The mastoid emissary connects the sigmoid sinus with the occipital or posterior auricular vein. The occipital emissary connects the torcular with one of the occipital veins. The parietal emissary is situated at the vertex of the skull, and connects the superior longitudinal sinus with one of the scalp veins. The condyloid emissary passes from the lower part of the sigmoid sinus, through the posterior condyloid foramen to the vertebral vein.

3. Through a venous ring which surrounds the hypoglossal nerve in the anterior condyloid foramen, and connects the occipital sinus with the upper part of the spinal plexus and the vertebral vein.

4. Through the carotid sinus which joins the cavernous sinus with the internal jugular vein.

5. Through vessels passing through the foramen ovale, the foramen spinosum, the middle lacerated foramen and the foramen of Vesalius which join the cavernous sinus to the pterygoid plexus. The pterygoid plexus empties into the internal and external jugular veins.

6. Through the cavernous sinus which communicates through the inferior ophthalmic vein with the pterygoid plexus.

7. Through the cavernous sinus which communicates through the superior ophthalmic vein, via the nasal vein, with the facial vein.

8. Through the superior longitudinal sinus which communicates through the middle meningeal vein with the pterygoid plexus.

9. Also, there is sometimes a communication between the superior longitudinal sinus and the frontal vein.

When there are infected thrombi within the sinuses, bacteria enter the general circulation principally through the internal jugular veins. But they may also enter through any of the communicating veins described above. Consequently ligation of the internal jugular vein does not shut off all the communicating channels between the infected clot and the general circulation.

Course of the Blood Stream within the Sinuses

The course of the blood stream within the venous sinuses of the skull, is, in general, from before backward, and from above, downward.

There are two main systems of sinuses, an upper and a lower. In the upper system, the blood flows from the upper and anterior part of the skull through several channels to the region of the torcular, and thence through the lateral sinuses to the internal jugular veins in the neck. In the lower system, the blood flows from the lower part of the skull and the orbits, along the base of the skull to the lower part of the lateral sinuses and the jugular bulb, and thence to the internal jugular veins. The upper system of sinuses consists of three branches. The uppermost of these is formed by the superior longitudinal sinus, the middle one by the inferior longitudinal sinus and the straight sinus, and the lower one by the occipital sinus. These three branches terminate at the torcular, where they empty into the lateral sinuses.

The lower system is composed of the cavernous sinuses, the circular sinus, the sphenoparietal sinus, and the superior and inferior petrosal sinuses. The blood stream, after passing back through the cavernous sinus, is split into two columns, one reaching the knee of the sigmoid sinus through

the superior petrosal sinus, and the other reaching the jugular bulb, through the inferior petrosal sinus.

Thrombi situated in various portions of the dural sinuses give rise to changes in the direction of the blood stream within the sinuses.

When there is a thrombus in the jugular bulb, the blood, after passing through the lateral sinus of the affected side, passes through the mastoid emissary and posterior condyloid veins to the occipital and vertebral veins. From the occipital vein, it passes through the external jugular to the subclavian vein. From the vertebral vein, it passes to the innominate vein.

The blood in the inferior petrosal sinus will flow back to the cavernous sinus, and then through the circular sinus to the opposite cavernous, and so through the opposite internal jugular vein. Some of the blood from the cavernous sinus will flow back through the superior petrosal to the lateral sinus, where it will escape through the mastoid and condyloid emissaries.

A thrombus in the sigmoid sinus, extending from a point above the entrance of the superior petrosal sinus to the bulb, will cause the blood to pass partly through the opposite lateral sinus to the opposite internal jugular vein, and partly through the occipital sinus and marginal sinus to the opposite internal jugular vein.

If the thrombus is situated in the lower part of the sigmoid sinus, below the entrance of the superior petrosal sinus, and not involving the jugular bulb, the blood, in addition to passing through the opposite lateral sinus and the occipital sinus, will flow backward through the superior petrosal sinus of the same side to the cavernous sinus. From here, it will pass through the inferior petrosal sinus and the jugular bulb to the internal jugular vein.

When the thrombus involves the entire lateral sinus, up to, but not including the torcular, the blood passes from the superior longitudinal sinus through the opposite lateral sinus. The blood from the cavernous sinus

passes through the circular sinus to the opposite cavernous, and then through the opposite petrosals to the sigmoid sinus and bulb.

If the thrombus in the lateral sinus extends beyond the torcular, the blood from the superior and inferior longitudinal sinuses passes through the meningeal veins which connect these sinuses with the cavernous sinuses. From the cavernous sinuses, the blood passes through the petrosals of the side on which the lateral sinus is not occluded. The superior longitudinal sinus also pours some of its blood into the veins of the scalp through the parietal emissary.

In thrombosis of the inferior petrosal sinus, the blood passes from the cavernous sinus through the superior petrosal to the lateral sinus.

In thrombosis of the superior petrosal sinus, the blood passes from the cavernous sinus through the inferior petrosal to the jugular bulb.

In thrombosis of both petrosals, with thrombosis of the cavernous sinuses of both sides, the blood flows from the ophthalmic veins through communicating vessels to the pterygoid plexus, and from there to the facial vein. A portion of the blood would also flow upward through the vena fossae Sylvii and the superior cerebral vein to the superior longitudinal sinus, and then through the lateral sinuses to the internal jugular veins.

When there is a thrombus in one cavernous sinus, the blood flows from the ophthalmic veins through the pterygoid plexus to the facial vein.

A thrombus of both petrosals, without an involvement of the cavernous sinus, causes the blood to pass from the cavernous sinus through the circular sinus to the opposite cavernous.

When, at operation, a thrombus has been removed from the sigmoid sinus, and a free return flow of blood is obtained from the lower end of the sinus, this flow may come from the internal jugular vein. But it may also come from the inferior petrosal sinus or the posterior condyloid vein, even when there is a clot in the lower part of the bulb or in the internal jugular vein.

The blood in the internal jugular vein is subjected to a marked aspiratory effect by the negative pressure which occurs in the chest during inspiration. This can be very plainly seen during operation on the jugular vein. When the jugular vein is exposed, it is seen to collapse during each inspiration, and to fill up during each ex-spiration. As the venous sinuses in the cranial cavity are rigid walled tubes which cannot collapse, it would seem that they would be emptied during each inspiration. But if this occurred, there would result an emptying of the cerebral veins during each inspiration, and an emptying of the small cerebral arterioles, with the occurrence of a temporary anemia of the brain. This would give rise to cerebral symptoms.

According to Macewen, there is a mechanism in the venous sinuses, which prevents their being emptied during inspiration. As a result of this mechanism, the venous sinuses are constantly full, and there is a steady, even flow through them, at all times.

The regulation of the blood stream in the sinuses depends upon the following factors:

1. The blood stream in the superior longitudinal sinus is retarded because the middle and posterior cerebral veins enter this sinus obliquely forward, that is, against the direction of the blood stream.

2. The blood stream in the superior longitudinal sinus splits at the torcular and bends at right angles to continue in the lateral sinuses. This results in a slowing of the blood stream.

3. In the lateral and sigmoid sinuses, the blood stream has a very tortuous course.

4. At its lower end, the sigmoid sinus curves sharply upward, and narrows down at its entrance into the jugular bulb.

5. The jugular bulb lies at a higher level than the lower part of the sigmoid sinus, and it is considerably wider than the sigmoid. It acts as a reservoir, and causes a considerable slowing of the blood current.

6. The jugular bulb is narrow at its outlet. This narrowing is increased by the other structures which pass through the jugular foramen, namely, the ninth, tenth and eleventh cranial nerves, and the inferior petrosal sinus.

7. The collapse of the walls of the internal jugular vein prevents suction of the contents of the bulb, to a certain extent.

8. The digastric and stylohyoid muscles pass along the outer side of the internal jugular vein at its upper end. When they contract, they exert a slight pressure on the walls of the vein.

As a result of all these factors, the aspiratory effect of inspiration upon the blood stream in the dural sinuses is neutralized, and there results an even, steady blood flow within the skull.

Chapter III

ETIOLOGY AND PATHOLOGY

Chapter III

ETIOLOGY AND PATHOLOGY

THE clinical picture of otogenic general sepsis may be caused by a number of different pathological conditions. In the vast majority of cases, however, it is caused by a thrombophlebitis of one or more of the dural venous sinuses. A similar clinical picture may occur with inflammation of the wall of one of the sinuses without thrombus formation. It may also occur with phlebitis or thrombosis of the veins in the middle-ear mucosa, or with infection in the venous channels in the mastoid process.

The clinical picture does not always parallel the local pathological lesion. The clinical picture may be that of an acute foudroyant sepsis, while the sinus may show slight or no pathological changes. On the other hand, there may be an extensive thrombus in the sinus, with no symptoms of general sepsis. In the former class of cases the infection is so severe that the patient succumbs before the sinus can throw up any protective barriers. In the latter class of cases, the thrombus in the sinus has succeeded in preventing the infective process in the temporal bone from invading the general circulation. A thrombus is a protective effort on the part of the system to prevent infection on the outside of a blood vessel from invading the general circulation. There are cases which show all gradations between these two extreme types.

In the vast majority of cases of otogenic general sepsis, there is an inflammation of the sinus wall, and more or less thrombotic material within the sinus.

Formation of a Thrombus

A thrombus is a mass of more or less solid consistency, which causes partial or complete occlusion of the vessel in which it lies, and which is formed

in situ, during life, from the constituents of the blood. It differs from an embolus, in that the latter is not formed in situ, but is carried by the circulation to the place where it lodges. Thrombi may occur in the heart, veins, arteries or capillaries, but they are most common in veins.

Thrombi are composed of fibrin, red and white blood cells and blood platelets. Most thrombi have a definite histological structure. A thrombus usually consists of a head, a neck and a tail. The head is usually grayish white in color, the neck is mixed grayish and red, while the tail is red. The head is the first portion of the thrombus to be formed, the neck is the intermediate portion, and the tail is formed last.

John Hunter, in 1793, believed that a thrombus was an inflammatory exudate, formed from the inflamed vessel wall, that is, "an extravasation of coagulable lymph."

Virchow believed that mechanical conditions of the blood stream were the important factors in causing thrombosis. He found that there were no inflammatory changes in the vessel wall, in many cases of thrombosis.

Blood Platelets. Eberth and Schimmelbusch, in 1888, were the first to recognize the important rôle played by the blood platelets in the formation of a thrombus.

Hayem, in 1877, first gave a complete description of blood platelets, and placed them on a firm histological basis as distinct elements of the blood.

Bizzozero, in 1882, first used the name blood platelets. He was the first to observe them in the living vessels. He saw them in the mesenteric vessels of the guinea-pig. He thought that they were important factors in the coagulation of blood.

It was determined by Wright, that the blood platelets are formed from the giant cells in the spleen and bone marrow.

Blood platelets are flat or slightly convex disc-like bodies from 1 to 2μ in diameter, that is, about one quarter of the diameter of red blood cells.

They are homogenous or slightly granular, and are devoid of color. When seen in profile, they have the appearance of small rods (Fig. 72). There are 400,000 to 600,000 blood platelets in 1 cu. mm. of blood. The proportion of

FIG. 72. A. BLOOD PLATELETS. B. RELATIVE SIZE OF RED BLOOD CELLS AND BLOOD PLATELETS. C. BLOOD PLATELETS UNDERGOING STELLATE CHANGE. D. BEGINNING CLUMPING OF BLOOD PLATELETS. E. CLUMPING OF BLOOD PLATELETS WITH BEGINNING DEPOSITION OF FIBRIN.

blood platelets to red blood cells is about 1 to 10. They lose their form within a minute or two after blood is shed. If the blood is examined ten to fifteen seconds after it is shed, the platelets are seen to be perfectly round. After a minute, their edges become notched, they shrink and become star-shaped. They become more granular and more refractive to light. They soon begin to adhere to each other, and in fifteen to twenty minutes they are formless masses of adherent granular material.

Eberth and Schimmelbusch observed thrombus formation in the small vessels in the omentum of dogs, during life. A portion of the omentum was placed on the stage of a microscope, immersed in a warm saline bath. On examining a moderately small blood vessel, they observed that it was not evenly filled with blood cells. In the center was a wide red stream, and at the periphery, a lighter thin zone of plasma (Fig. 73). In the axial stream, the flow was so rapid that the individual red cells could not be distinguished. In the marginal plasma zone, single leucocytes were seen which travelled more slowly than the axial stream. The marginal portion of the stream travelled more slowly than the central portion on account of its friction with the vessel wall. It was determined experimentally, that bodies of the greatest specific gravity travel in the center of the stream, and the lightest particles

travel at the periphery. White blood cells are lighter than red blood cells. Platelets are about as heavy as red cells, and travel in the center of the blood stream.

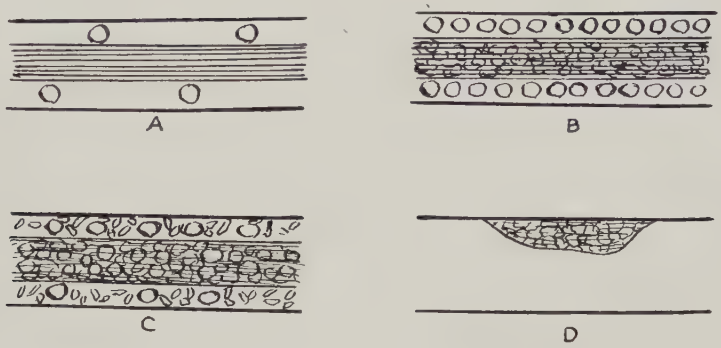

FIG. 73. A. NORMAL BLOOD STREAM IN MEDIUM-SIZED VESSEL. DARK RED STREAM IN CENTER, AND CLEARER PLASMA ZONE AT PERIPHERY CONTAINING A FEW LEUCOCYTES.

B. BLOOD CURRENT SLOWED. LEUCOCYTES IN MARGINAL ZONE INCREASE IN NUMBER.

C. BLOOD CURRENT SLOWED STILL MORE. BLOOD PLATELETS BEGIN TO APPEAR IN MARGINAL ZONE IN ADDITION TO THE LEUCOCYTES.

D. PLATELET THROMBUS ADHERENT TO WALL OF VESSEL. (*After Eberth and Schimmelbusch.*)

When the blood stream was slowed, the leucocytes in the marginal zone increased in number. As the current was slowed still more, blood platelets began to appear in the marginal zone.

Where there were irregularities in the lumen of the blood vessels, such as narrowing or widening, or pointed projections into the lumen, whirlpools occurred in the blood stream. Here the leucocytes and blood platelets also appeared in the marginal plasma zone of the blood stream.

When the vessel was compressed by a blunt glass rod, a stasis occurred in the blood stream between the compressed point and the nearest collateral. In this area of stasis, all the elements of the blood were at first mixed together. After one or two minutes, the red cells became more compact, and left clear areas of plasma at the margin of the blood column. The clear areas

filled with leucocytes and platelets. A wave-like movement was transmitted to the contents of the vessel in which there was stasis, by the blood current in the adjacent normal vessels. This caused the blood platelets to be thrown against the wall of the vessel and to stick to the wall and to each other. The platelets soon showed pointed margins, and eventually formed a platelet thrombus. Similar results were obtained by damaging the vessel wall with chemical agents. Sometimes leucocytes were caught in these masses of platelets. Less often red cells were entangled. Blood platelets are the important factors in the formation of these thrombi. White and red cells are only accidental inclusions.

Platelet thrombi occur only when the rapidity of the blood current is very much retarded, or when the stream is disturbed by whirlpools. In the normal blood stream, the platelets are protected by being carried in the center of the stream, so that they cannot touch the wall of the blood vessel. When the platelets are thrown against the wall of the blood vessel, especially when the wall is injured, a platelet thrombus is formed. This type of thrombus is called a conglutination thrombus, because it results from the conglutination or agglutination of the platelets to each other and to the vessel wall.

In most thrombi, in addition to the massing of the blood platelets, there is a deposition of a certain amount of fibrin. The fibrin is deposited a short time after the platelets have become adherent to the vessel wall. The fibrin has no direct connection with the blood platelets. When blood is drawn, fibrin deposit begins after one or two minutes. The formation of fibrin is a crystallization process. The fibrin fibers are composed of spindle-shaped crystalline needles 2 to 5μ long. They are pointed at both ends (Fig. 74). They gradually unite and form the typical fibrin threads.

Formation of Fibrin. The blood plasma contains, in solution, a proteid material called fibrinogen. It also contains a nucleo-proteid called

prothrombin. When blood is shed, the disintegrating blood platelets and leucocytes liberate a fibrino-plastic substance called thrombokinase. The thrombokinase, in combination with the calcium salts in the blood, converts

FIG. 74. FIBRIN CRYSTALS. (*After Eberth and Schimmelbusch.*)
A, low magnification; B, high magnification.

the prothrombin, which is an inactive substance, into the active thrombin. The thrombin combines with the soluble fibrinogen, and produces the insoluble fibrin.

STRUCTURE OF THROMBI. If a longitudinal section of the head portion of a thrombus is examined under the microscope, it is seen to consist of a framework of beams or trabeculae, composed entirely of agglutinated masses of blood platelets. The beams are covered by a layer of leucocytes. Red blood cells and fibrin fill the spaces between the beams. The beams are grouped into systems which are parallel to each other. There are side beams, which are attached obliquely to the main systems. The nearer they come to the head of the thrombus, the thicker they are, and the smaller are the interspaces. The beams are at right angles to the vessel wall. Some of them are oblique to the vessel wall. They are bent in the direction of the blood stream. The ends of these beams appear on the free surface of the thrombus in the form of ribbed markings. These ribbed markings are similar in appearance to the ripples on the sand of the seashore, left by ebb-tide.

The platelet thrombus is formed in the flowing blood stream. Aschoff showed that the platelets are deposited in this way when there is a slowing of the blood stream with the formation of eddies and whirlpools, due to

roughening of the vessel wall and to irregularities in the shape of the blood vessels. He showed the analogy of this occurrence to that which happens in a river, when the water contains particles of sand. Where there is a change

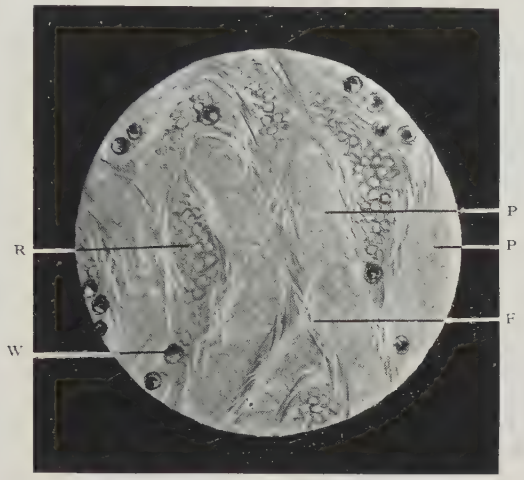

FIG. 75. MIXED THROMBUS (×300.)

F, fibrin; P, beams consisting of blood platelets; R, red blood cells; W, white blood cells.

in the current, that is, where there is a widening or deepening of the stream, the particles of sand are deposited on the bottom, in exactly the same form as in the platelet thrombus. He reproduced this condition by interposing dams and weirs in the course of a stream of water, in which particles of sawdust were suspended. At the points where the current of water was disturbed by the dams and weirs, the particles of sawdust were deposited in the typical ripple formations.

The beams are covered by leucocytes, because, when the blood stream is slowed, the lighter leucocytes pass to the edge of the blood current. The red blood cells are entangled between the beams. Fibrin is deposited between the beams (Fig. 75).

As long as the blood current flows past the beams, new beams of platelets are deposited. When the lumen of the vessel is filled by this material,

the primary thrombus reaches its completion. The blood column up to the next branch of the vessel becomes stationary, and this column of blood undergoes clotting. This forms the red tail of the thrombus. The red thrombus is just like the clot which forms in shed blood, and consists of an irregular mass of red and white blood cells, platelets and fibrin. The clotting in the tail of the thrombus occurs as a result of the liberation of ferments from the death of the platelets and leucocytes in the head of the thrombus. The red thrombus is formed not by the deposition of formed elements from the blood, as in the case of the white thrombus, but by the formation of fibrin from the blood. The red thrombus is a coagulation thrombus in contrast to the white thrombus which is a conglutination or agglutination thrombus.

Not all thrombi become obturating. Sometimes the thrombotic process stops before the entire lumen of the vessel is occluded. When the thrombus does not fill the vessel, it is called a parietal thrombus or a mural thrombus. Mural thrombi occur either in very virulent infections or in very mild infections. In the very virulent infections, the patient dies before the thrombus becomes obturating. In the mild infections, the thrombotic process is arrested before the thrombus becomes obturating.

Causes for Formation of Thrombi

The three most important factors in the causation of thrombosis are, (1) slowing of the blood stream, (2) injury to the endothelium lining the vessel wall, and (3) changes in the constitution of the blood.

Slowing or even stagnation of the blood current alone is not sufficient to cause thrombus formation. Baumgarten showed that when a vessel is double ligated, aseptically, in such a way that the endothelium is not injured, the column of blood between the two ligatures will remain fluid for several weeks. Whirling or eddying movements of the blood are more important than slowing of the current, as was shown by von Recklinghausen. There is

normally a slowing of the blood current in the venous sinuses of the skull as a result of the rigidity of their walls, the absence of muscular fibers in the walls, and the great width of their lumen as compared to the small size of the veins which empty into the sinuses. Whirlpools occur in many places in the sinuses, as a result of the tortuosity of their course. When there is a general sepsis, there is still greater slowing of the current, on account of the toxic effect upon the heart. The thromboses which occur so frequently after operations are due, in part, to the slowing of the blood current which results from loss of blood at the operation, weakening of the heart action from shock, and general prostration and confinement to bed.

The second important factor in the production of thrombosis is injury to the intima of the blood vessel. This injury has both a physical and a chemical effect. The physical effect occurs when the endothelium is injured, and the smooth glistening inner surface of the vessel is lost. The rough surface interferes with the even flow of the blood current, and also allows the blood platelets to adhere to the vessel wall. The chemical factor in the relationship between injury to the endothelium and the formation of a thrombus depends upon the fact that breaking down of the endothelial cells liberates thrombokinase. Thrombokinase is necessary for the formation of fibrin. It also seems to have the effect of making the blood platelets adhere.

The injury to the endothelium is usually caused by the destructive effect of the bacterial poisons in the vessel wall. The slowing of the blood current also tends to cause a breaking down of the endothelial lining, because the endothelium receives its nourishment from the blood in the lumen of the vessel and not from the vasa vasorum. Consequently, if the blood stream is markedly slowed, the endothelium does not receive sufficient nourishment to maintain its integrity.

That destruction of the endothelial lining of a vessel is not the only factor necessary to produce a thrombus is shown by the fact that thrombosis is very rare in extensive atheromatous disease of the aorta.

Changes in the constitution of the blood which favor thrombosis are increase in the amount of fibrinogen, prothrombin, or calcium salts in the blood. In order for a thrombus to occur, it seems that all three factors must act together, namely, slowing of the blood stream, injury to the endothelium, and changes in the constitution of the blood.

Bacteria in the wall of the vein play an important rôle in the production of thrombosis. Most cases of thrombosis of the cerebral sinuses are caused by an inflammatory process in and around the sinus wall, with bacterial infection. However, there are cases of so-called marantic thrombosis, in which there is no evidence of bacterial infection. This form of thrombosis occurs in long continued wasting diseases in children, such as protracted diarrheas and phthisis. There is slowing of the blood current due to feeble heart action. The resultant anemia is accompanied by an increase in the blood platelets. According to Haward, anemic blood is deficient in oxygen and overloaded with carbonic acid, by reason of its poverty in hemoglobin. An increase of carbonic acid in the blood increases its coagulability. A. E. Wright relates a case of hemophilia, in which hemorrhage was arrested by the inhalation of carbonic acid gas.

Marantic thrombi occur most commonly in the superior longitudinal sinus. There are several anatomical factors which predispose to the occurrence of marantic thrombosis: (1) the stiff immobile walls of the sinus with its lack of elastic fibers and absence of a muscular wall; (2) the angularity of the lumen of the sinus; (3) the relatively large size of its lumen compared to the smallness of the veins which open into it; and (4) the presence of Pacchionian bodies and the network of septa in the sinus. All of these conditions cause a slowing of the blood stream.

Other predisposing factors, in addition to wasting diseases, are thermic insults, such as sunstroke, the pressure of tumors, and trauma to the head without fracture.

When bacteria are present in the sinus wall, the thrombotic material covers the infected area, and attempts to prevent infection of the blood stream by the bacteria. In other words, in cases of infective sinus phlebitis, thrombosis is a protective process. It is not always successful in this effort. When the thrombus is overpowered by the infection, it breaks down, bacteria enter the general circulation, and general sepsis results.

When the infective organism is very virulent and the resistance of the patient is poor, bacteria pass through the sinus wall directly into the blood stream, without the formation of a thrombus. In these cases, the patient dies before a thrombus has had time to form.

Bacteria as a Cause of Thrombosis. The action of bacteria in producing a thrombus is three-fold:

1. They cause a breaking down of the endothelium lining the sinus. Roughening of the vessel wall is an important factor in the production of thrombosis. When there is a wound in the endothelial lining of the vessel, there is a disturbance in the interchange of the plasmatic current between the vessel wall and the blood stream. This disturbs the normal blood current.

2. The bacteria elaborate toxic material in the vessel wall. These toxins pass through the wall into the blood and produce certain chemical changes in the blood. These chemical changes favor thrombus formation. The toxins also have a destructive effect upon the blood corpuscles.

3. The toxemia resulting from the sinus infection has a deleterious effect upon the heart muscle and the vasomotor centers. This results in slowing of the blood stream, which is also an important factor in the production of thrombosis. Slowing of the blood stream, in otogenic sinus thrombosis, may also be caused by compression of the sinus by a perisinous abscess, by

inflammatory thickening of the sinus wall, or by the tamponade which is sometimes employed after a mastoid operation, especially when there has been an accidental injury to the sinus.

Methods in Which Infectious Thrombosis Occurs. Aschoff describes three methods in which infectious thrombosis may occur:

1. Bacteria progress through the sinus wall, producing a primary phlebitis with a secondary deposition thrombus;

2. A non-infected thrombus may occur in the sinus from mechanical causes. This thrombus may become infected by bacteria in the blood stream. Secondary inflammation of the sinus wall then occurs.

3. A thrombus may occur in one or more of the venous radicles of the sinus, with an extension of the thrombus into the sinus.

A thrombus in the sinus may grow in two ways:

1. When the sinus is blocked by the primary thrombus, there is stasis of the blood column proximal to the occluded area, with clotting of this column of blood.

2. When the phlebitic process extends along the sinus wall, the thrombus formation extends along with the inflammatory process in the sinus wall.

Growth of the thrombus by the first method is in the direction of the blood stream. Growth of the thrombus by the second method may be either in the direction of the blood stream or against the direction of the blood stream.

The growth of a thrombus is not always continuous. The presence of two or more discontinuous thrombi in the sinus is not uncommon. This may occur in one of several ways. A thrombosed dural vessel may open into the sinus some distance from the thrombus in the latter. The thrombus from the dural vessel may grow into the sinus and form a thrombus which is not connected with the original thrombus in the sinus.

The several thrombi in the sinus may each be due to extension of thrombi from vessels which open into the sinus.

Bacteria from the thrombus may get into the circulation and lodge in some other location, for example, in the jugular bulb where the anatomical conditions are favorable for multiplication of the bacteria and the formation of a thrombus.

The infection in the sinus wall may spread unevenly, so that there are areas here and there, in the sinus wall, where the inflammation is less intense. In the less inflamed areas, the disease may not reach the intima of the sinus. The thrombus may form only in those areas where the endothelium is destroyed, while the areas where the endothelium is normal are free from thrombotic growth.

There may be complete absorption of the central part of a thrombus, while the ends go on to organization. In this way a picture results, which looks like discontinuous thrombi.

Bacteria may pass from the original thrombus embolically, through vasa vasorum, and produce inflammation of the sinus wall with thrombus formation, in another location.

Organization of the thrombus begins within a few days after the thrombus has been formed. Leucocytes and connective tissue wandering cells enter the thrombus from the vasa vasorum in the wall of the sinus. These cells remove the products of the breaking down of tissue in the thrombus. From the endothelium and the subendothelial connective tissue, spindle-shaped connective tissue cells and vascular sprouts grow into the thrombus. The endothelium of the intima hypertrophies, and wherever there is a space between the vessel wall and the thrombus, the endothelium crosses over to the thrombus and grows into all the clefts of the latter, forming endothelial lined cavities. Portions of the thrombus are absorbed, the fibrin remaining for a considerable time. The spindle-shaped cells elongate and become

transformed into connective tissue fibers. The thrombus is gradually transformed into connective tissue, containing many new-formed blood vessels. The vascular sprouts which grow into the thrombus connect with the spaces in the thrombus which are lined by endothelium. As the thrombotic tissue is absorbed, these spaces increase in size, and the circulation within the sinus is partly or entirely re-established. This is called recanalization of the sinus.

When a thrombus is caused by an infection in the sinus wall, the thrombus itself soon becomes infected. Bacteria enter the thrombotic mass, and cause softening and breaking down of its tissue. The breaking down may occur either in the center or at the ends of the thrombus. When it occurs near the ends of the thrombus, particles of infected thrombotic material are broken off and carried into the circulation. They may lodge in some distant part of the body.

Organization of a thrombus may occur whether the thrombus is infected or not. Broken down areas are often seen side by side with areas showing reparative changes.

Bacteria usually enter the thrombus from the vessel wall. But they may be caught up from the circulation. In the latter case, the wall of the sinus may become infected secondarily from the thrombus. Sometimes an abscess forms in the thrombus and perforates through the outer sinus wall, producing a perisinous abscess.

It is unusual for hemorrhage to occur as the result of spontaneous perforation of the sinus wall in cases of sinus phlebitis, because the lumen of the sinus is usually occluded by thrombotic material, before the perforation in the sinus wall occurs. But occasionally spontaneous hemorrhage does occur from a sinus which is inflamed. The writer observed 3 such cases, in which the sinus was uncovered during the performance of a mastoidectomy. The sinus wall looked normal at this time. Some time after the operation,

(in the writer's cases from one to two weeks) an area of necrosis appeared in the outer wall of the sinus. This area broke down and was followed by very free bleeding. In 1 case, this was followed by symptoms of general sepsis with the occurrence of metastases. Operation on the sinus showed no evidence of thrombosis. In the other 2 cases, the wound in the sinus wall healed without the occurrence of septic symptoms. The infective organism in all 3 cases was a streptococcus. There was probably some peculiarity in the type of streptococcus, however, which caused it to produce necrosis of tissue rather than thrombosis.

Accidental injury to the sinus or emissary during a mastoid operation may or may not be followed by thrombosis. An aseptic tear in the sinus wall never results in thrombosis. But when the mastoid cavity is infected, thrombosis may occur, especially when pressure is used to control the bleeding from the sinus, and when some infected bone is allowed to remain in contact with the sinus wall. In some of the cases, the wall of the sinus is torn because it is already weakened by inflammatory disease. A mural thrombus may already have been present at the time of the injury. The thrombus may become obturating after the injury.

Results of Thrombosis

1. The throwing off of particles of thrombus and bacteria into the circulation, with or without the occurrence of metastases.

2. The occurrence of stasis of blood beyond the thrombus, when the thrombus is an obturating one.

3. Edema of the tissue in which the venous radicles which empty into the thrombosed sinus lie.

4. Hemorrhage into the tissue in which the venous radicles lie.

5. Inflammation of the tissue about the thrombosed sinus. This results in periphlebitis, perisinous abscess, subdural abscess, brain abscess and meningitis.

Variations in Results of Thrombosis. These depend upon the following factors:

1. The completeness and the rapidity of the closure of the sinus by the thrombus.

2. The nature of the thrombus, whether bland or infected. Bland thrombi do not cause inflammatory changes in the tissues about the sinus.

3. The condition and the type of the surrounding tissue, that is, its vascularity, its resistance to infection, etc.

4. The speed and the completeness of the establishment of collatera circulation.

A *metastasis* is the occurrence of a new similar focus of disease in some other location than the original focus.

Embolism is the carrying away and deposition of the contents of the blood stream into some other part of the blood stream. The material which is transported through the blood stream is an embolus. The embolus may consist of a piece of thrombus or of a clump of bacteria. The result of a bacterial embolus is usually the formation of a metastasis. Bacteria do not multiply in the circulating blood, as they are rapidly destroyed by the bactericidal action of the blood. This is the reason why metastases do not occur in every case of sinus phlebitis. If the infecting organism is very virulent, however, and in many cases just before death, bacteria do multiply in the blood. The septic symptoms of a general sepsis are produced by a continuous or intermittent invasion of the blood stream by bacteria which are released from the thrombophlebitic focus.

An embolus which consists of a fragment of a thrombus in one of the cerebral sinuses passes to the right heart, and then through the pulmonary artery, and lodges in one of the small arterioles or capillaries of the lung. If the embolus is very minute, especially if it is composed of bacteria alone, without a surrounding envelope of thrombotic material, it may pass through

the pulmonary capillaries and lodge in the peripheral circulation. When there is a patent foramen ovale in the heart, emboli of large size may pass into the peripheral circulation without passing through the lung.

There may be a retrograde transport of emboli from the sinus into some part of the peripheral circulation, when there is a reversal of the blood current in the sinus. The possibility of retrograde transport of emboli was first proven experimentally by Virchow. He placed mercury in a vein, and found particles of the mercury in centrifugally situated veins. Von Recklinghausen found that when the normal negative pressure in the chest is changed temporarily to a positive pressure, as in violent coughing or difficult breathing, retrograde transport of an embolus in a vein may occur. When there is occlusion of the lateral sinus by a thrombus, there may be retrograde transport of a piece of thrombus, as the result of a reversal of the blood current in the petrosal sinuses and cavernous sinus, or in the superior longitudinal or straight sinus. When there is an obturating thrombus in the lateral sinus, there is a stagnation in the collaterals of the sinus and bulb, with an increase in the blood pressure in the collaterals. As a result of the absence of valves, there results a retrograde current in these vessels in order to equalize the blood pressure. This retrograde current is directed toward the opposite jugular or toward the spinal and vertebral venous plexuses. The embolus which is thus carried into the peripheral circulation by retrograde transport may result in metastases in the brain, meninges, extremities or abdominal organs.

Occlusion of a small artery by an embolus may give rise to an infarct. There are two kinds of infarcts, anemic and hemorrhagic. An anemic infarct is a pure tissue necrosis, due to lack of blood supply. A hemorrhagic infarct is a tissue necrosis with hemorrhages. The shape of the infarct always corresponds to the area of branching of the artery. It is usually wedge-shaped or pyramidal. An infarct occurs only in an organ in which the arterial anasto-

moses are insufficient to provide collateral circulation. The blood pressure in the veins adjacent to the infarct is higher than in the vessels of the infarct itself. Consequently there is a current backward from these veins into the infarct. As the capillary walls are necrosed, there is hemorrhage into the area of the infarct. When bacteria are lodged in the piece of thrombus which is caught in a small artery, an infarct is formed first, which later breaks down and forms a metastatic abscess.

Inflammation of the Sinus Wall

In otitic sinus thrombosis, the condition is not a thrombosis but a thrombophlebitis. The inflammation of the sinus wall is a very important part of the pathological picture. The inflammation usually starts in the outer sinus wall. It may extend to the inner or visceral sinus wall either by continuity from the outer wall, or from the thrombus, when the latter becomes completely obturating.

The outer sinus wall usually shows the picture of acute exudative inflammation (Fig. 76). The perivascular connective tissue and the fibrous wall of the sinus are thickened. The lymph spaces and the fibrous tissue itself contain a finely granular exudate, rich in fibrin and leucocytes. The vasa vasorum are dilated, full of blood, and are surrounded by dense masses of round cells. Sometimes there are thrombi in the vasa vasorum. The connective tissue cells show evidence of necrosis, their nuclei taking the stain very poorly. The subendothelial connective tissue layer of the intima is infiltrated with exudate and thickened. The endothelium may be separated in places from the underlying tissue and adherent to the thrombus. In some places, there may be two or more layers of endothelium, but in most places the endothelium is wanting altogether. Where the endothelium is present, many leucocytes are seen passing through it.

According to Esch, the type of inflammation in the sinus wall corresponds to the type of inflammation in the bone overlying the sinus. The three principal types of inflammation in the sinus walls are the *plastic*

FIG. 76. THROMBOPHLEBITIS WITH ORGANIZING MURAL THROMBUS AND PERISINOUS ABSCESS.

B, bacteria in sinus wall; E, endothelium; P, perisinous abscess; S, thickened sinus wall, with round-celled infiltration; T, organizing thrombus.

exudative type, the *proliferative type* and the *necrotic type*. In the first class of cases, the bone overlying the sinus, and the sinus wall, are covered and infiltrated with pus cells and fibrinous exudate. In the second class of cases, the bone overlying the sinus is broken down and replaced by vascular granulation tissue. The sinus wall is thickened and infiltrated with round cells, and contains newly formed blood vessels and connective tissue cells. In the third class of cases, there are no reparative changes. There is necrosis of the overlying bone with no evidence of reaction. There is pus between the bone and the sinus wall. There is necrosis of the sinus wall, with no evidence of proliferation. This class of cases occurs frequently with scarlet fever and other severe types of infection, especially those caused by anerobic bacteria. The changes in the thrombus usually correspond to the changes in the sinus wall.

Bacteria are usually present in the sinus wall. In the early stages of thrombophlebitis, most of the bacteria are found in the outer layers of the sinus wall. They diminish in number toward the lumen of the vessel. Sometimes there are no bacteria in the innermost layers of the sinus wall and in

the thrombus. The bacteria are found mostly in the lymph vessels and lymph spaces in the sinus wall.

When the visceral wall of the sinus is involved, the changes in it are similar to those in the outer wall.

At a later stage, numerous small abscesses may appear in the wall. These coalesce and form a single large abscess, which breaks through externally and into the lumen of the sinus, leaving a hole in the sinus wall. This usually occurs when the thrombus has undergone purulent change. Then the contents of the sinus drain through this opening. When the hole is located in the outer sinus wall, a perisinous abscess results. When the hole is located in the inner sinus wall, a subdural abscess, brain abscess or meningitis results.

Sometimes the inflammatory changes are limited to the inner sinus wall, the outer wall remaining normal. Streit in experiments on dogs found that occasionally, when an infected tampon was placed over the sinus, the outer sinus wall remained normal, but a pachymeningitis interna resulted, which extended to the visceral wall of the sinus. This gave rise to a thrombus in the sinus. There is little doubt that this mode of infection occurs sometimes in the human being.

When the infection is not very virulent, the changes in the sinus wall are neither exudative nor necrotic, but proliferative. There is thickening of the sinus wall due to the formation of new connective tissue.

In most cases, the inflammatory process is primary in the sinus wall, and the thrombus is involved secondarily. Sometimes, however, the thrombus is formed before there are any changes in the sinus wall, and the latter becomes infected secondarily from the thrombus.

Infectivity of the Thrombus

The infectivity of the thrombus varies very much in different cases. Not infrequently, the thrombus is sterile. When bacteria are present in the

thrombus, they are usually more numerous in the central portion. Older portions of the thrombus are often bacteria-free or poor in bacteria, while the more recent portions contain many bacteria. The conical ends of an obturating thrombus are often sterile, but they may be rich in bacteria. The macroscopic appearance of a thrombus gives no idea of its infectiousness, except when it is broken down. The presence of infection in a thrombus is no bar to organization of the thrombus. Microscopic examination of thrombi often shows breaking down in one portion and organization in another.

In most cases of sinus thrombosis, the inflammatory tract can be followed macroscopically from the middle ear to the thrombus. There is necrotic bone in the mastoid which leads directly to the sinus wall. This is especially likely to occur in chronic middle ear suppuration, when cholesteatomatous material lies in contact with the sinus wall. In acute mastoiditis, there is often destruction of the inner table over the sinus, with an accumulation of pus between what is left of the inner table and the sinus wall. The outer sinus wall is covered with granulations, and the wall is inflamed up to the intima. In some cases, however, no such macroscopic tract is evident. There may be no breaking down of bone in the mastoid process and the outer surface of the sinus wall may appear perfectly normal. In such cases, the infection may reach the interior of the sinus through the small veins with which the mastoid is so richly supplied, or through one of the dural veins which has become diseased through contact with a diseased tegmen antri; or the thrombus may extend to the lateral sinus from one of the other cerebral sinuses. This is especially likely to occur from the jugular bulb. Extension of a pachymeningitis interna to the cerebral wall of the sinus may cause a sinus thrombosis. When there is a suppurative labyrinthitis, a saccus empyema may occur. As the saccus lies in the dura which forms the inner wall of the sigmoid sinus, saccus empyema may give rise to a thrombus in

the sigmoid sinus. Kramm reported such a case. Fracture of the skull is a rather uncommon cause of sinus thrombosis.

The more virulent the infective process in the middle ear and mastoid, the less likely are we to find definite macroscopic changes leading to the outer wall of the sinus, and the less marked is the thrombophlebitic process in the sinus itself. The thrombophlebitis is a protective effort on the part of the body. It is an attempt to prevent the circulation from being flooded with bacteria. If the infection is not too severe, and the body resistance is good, the effort is successful for a time, and sometimes permanently. The thrombus organizes, and the patient recovers, or the thrombus is removed by operation, and the patient recovers. We have no means of estimating how often a patient with sinus thrombosis recovers without operative interference. In most cases, when the resistance is not so good, the thrombus becomes infected and breaks down, and the circulation is overwhelmed by bacteria.

When the infection is very virulent, and the body resistance is poor, the sinus either makes a feeble attempt or no attempt at all to block off the circulation, and the bacteria pass unhindered through the sinus wall into the circulation, without the formation of a thrombus.

There has been considerable controversy for many years as to whether all cases of otogenic general sepsis are accompanied by the occurrence of sinus thrombosis. There is no longer any doubt that bacteria may enter the circulation from the ear without the intervention of a thrombus in one of the sinuses. The cases without thrombus formation are the rapidly fatal ones. Haymann showed, in his experiments on dogs and monkeys, that bacteria may pass through the sinus wall and enter the circulation without the presence of a thrombus in the sinus. He was able to demonstrate this histologically. A number of cases have been reported, where there was otogenic general sepsis with bacteriemia, in which a thorough post-mortem

examination with histological examination of all the sinuses failed to reveal evidence of a thrombus in any of the sinuses. In some of the cases, there may be thrombi in some of the small veins in the mucous membrane of the middle ear and mastoid, or in the dural veins. In an overwhelming majority of cases of otogenic general sepsis, however, there is a phlebitis of one or more of the sinuses which are in anatomical relation to the temporal bone, and some thrombus formation in one or more of the sinuses. The thrombus may be very small, and may not completely fill the lumen of the sinus, or there may be very extensive thrombosis involving many or all of the cerebral sinuses simultaneously.

According to Brieger, a thrombus may be present in the sinus, and still not be the source of the general sepsis. This is especially true of parietal thrombi. He states that "a parietal thrombus in the sinus can only be assumed when it is demonstrated; but even in cases which are positive anatomically, the proof of the presence of a parietal thrombus must be completed by the further proof that the thrombus is so situated that it took its origin from the ear, and is so constituted, that it can produce general sepsis and metastases." He believes, for example, that in cases of otogenic general sepsis, in which autopsy shows a small, partly organized parietal thrombus attached to the cerebellar wall of the sigmoid sinus, the thrombus is not the cause of the general sepsis. A thrombus of this kind is too insignificant and contains too few bacteria to keep up a general sepsis; and furthermore the thrombus is not an extension from the middle-ear suppuration, but is due to incision and tamponade of the sinus.

End-results of a thrombus in the sinus, which has not been interfered with surgically:

1. The patient may die of general sepsis before any changes occur in the thrombus.

2. The thrombus breaks down in the center, and the pus is evacuated through the outer wall of the sinus, so that the abscess within the sinus communicates with the perisinous abscess.

3. The pus within the thrombus breaks through the visceral wall of the sinus, and produces a subdural abscess, brain abscess or meningitis.

4. The thrombus organizes and results in an obliteration of the sinus.

5. Part or all of the thrombus becomes absorbed, with recanalization of the sinus.

Cause and Extension of Thrombus in the Different Sinuses

In the vast majority of cases, the thrombus is situated in the sigmoid portion of the lateral sinus, as this is the part of the sinus which is in close relation to the mastoid antrum. From here, the thrombus may spread either against the blood stream or with the blood stream. When the spread is against the direction of the blood stream, it may reach the torcular. Occasionally it extends into the superior longitudinal or straight sinuses. Rarely does it pass into the opposite lateral sinus. It may extend into the superior and inferior petrosals, and the cavernous sinuses. Occasionally it extends into the vena cerebri magna (Galeni) and the cerebral veins which flow into the latter. There may be extension into the arachnoidal veins over the occipital and parietal lobes.

The thrombus may extend in the direction of the blood stream, into the mastoid emissary, the anterior and posterior condyloid veins, the jugular bulb and the internal jugular vein. It may extend through the jugular to the superior vena cava. Sometimes it extends from the internal jugular, against the current into the facial and thyroid veins.

The primary site of the thrombus may be in any of the other sinuses, the lateral sinus remaining uninvolved, or becoming involved secondarily. Perhaps the most common site, next to the sigmoid sinus, is the jugular

bulb. The bulb is in close relation to the middle ear. When there is a dehiscence in the floor of the middle ear, the infection may travel uninterruptedly from the middle-ear mucosa to the membranous wall of the jugular bulb. This is especially apt to occur in children.

Budde found dehiscences in the floor of the middle ear in 4.3 per cent of skulls examined by him. Müller found dehiscences in 6.5 per cent of skulls examined. Körner found dehiscences in 3.3 per cent of skulls examined.

Even when there is a layer of bone of considerable thickness between the middle ear and the roof of the jugular fossa, the inflammatory process may travel in this way. This plate of bone frequently becomes necrotic, even when the mastoid cells are uninvolved. The passage of an inflammatory process through a physiological dehiscence to the jugular bulb was first macroscopically observed by Bürckner. It was first demonstrated in histological sections by Brock.

Wittmaack demonstrated the fact that isolated bulb thrombosis may occur as the result of transmission through small veins which pass through the bony floor of the middle ear. He found that in the normal development of the temporal bone, there is a temporary stage, in which vascular sprouts from the dura grow into the loose subepithelial tissue of what is going to develop into pneumatic cells; so that there is a direct vascular communication between the dural periosteum and the mucous membrane of the middle ear. In normal development, this stage quickly passes. The thick vascular subepithelial tissue becomes the thin non-vascular mucoperiosteal covering, with obliteration of the vascular connections. But in cases of pathological pneumatization of the temporal bone, there is frequently a definite persistence of this vascular connection. The thick hyperplastic mucous membrane which persists as the result of the developmental disturbance and inflammatory irritation, demands a persistence of the vascular supply, which under

normal conditions is only a passing stage. In this way, there exist during the entire lifetime of these individuals, abnormal anatomical conditions for the extension of inflammatory processes by way of preformed vascular

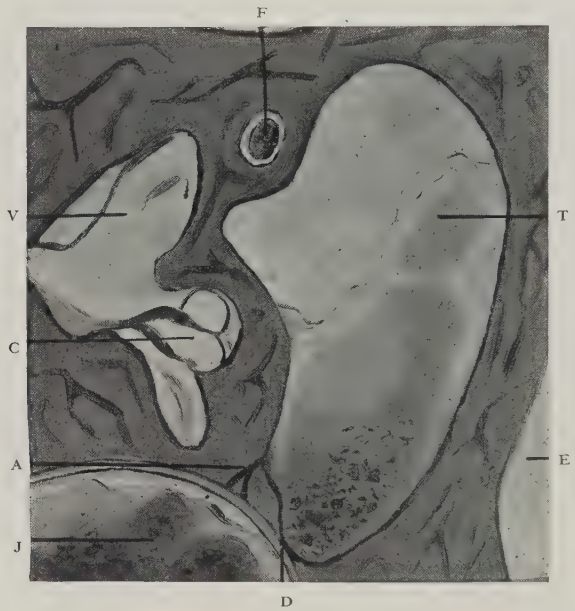

FIG. 77. VERTICAL SECTION THROUGH MIDDLE EAR AND JUGULAR BULB, SHOWING SPREAD OF INFECTION FROM MIDDLE EAR TO BULB VIA DEHISCENCE IN FLOOR OF MIDDLE EAR, AND VIA VEIN PASSING THROUGH FLOOR OF MIDDLE EAR.

A, infected vein passing from the middle ear to the jugular bulb; C, cochlea; D, dehiscence in floor of middle ear; E, external auditory canal; F, facial nerve; J, jugular bulb containing thrombus; T, middle ear containing inflammatory exudate; V, vestibule.

channels. These channels are much more frequently the pathways of extension, than the normal canals of exit of nerves, or fissures.

There are three principal locations where these vascular communications persist:

1. At the junction of the tegmen antri and tegmen tympani, in front of the convexity of the superior semicircular canal.

2. On the posterior surface of the petrous pyramid, above and lateral to the mouth of the aqueductus vestibuli.

3. On the floor of the middle ear.

The vessels passing through the posterior surface of the petrous pyramid may transmit infection from the middle ear to the sigmoid sinus. Infection along the vessels which pass through the floor of the middle ear results in bulb thrombosis. Wittmaack reported several cases of bulb thrombosis in which he was able to demonstrate histologically the spread of infection from the middle ear, through these vessels, to the jugular bulb (Fig. 77).

Infection sometimes travels from the middle ear along the canals for the auricular branch of the pneumogastric and the tympanic nerves, which emerge from the temporal bone on the lower surface of the plate of bone between the jugular fossa and the carotid canal.

Primary bulb thrombosis is sometimes traumatic in origin. The bulb may be injured in the course of a radical mastoid operation, or during a paracentesis of the drum-membrane, when the bulb pushes up the floor of the middle ear. In fracture of the base of the skull, there may be a fissure passing through the jugular fossa.

Quite frequently a row of mastoid cells lies just below the posterior wall of the external auditory canal, behind the descending portion of the facial nerve. One of these cells may break through the lateral wall of the jugular fossa, in suppurative disease of the mastoid, and give rise to a thrombus in the bulb (Fig. 78).

Leutert believes that primary bulb thrombosis may occur as the result of an indirect embolic process. He believes that bacteria sometimes pass from the mastoid through the diseased sinus wall into the blood currents and lodge in the bulb, where favorable conditions are present for their multiplication. These favorable conditions consist in the fact that the blood current from the sigmoid sinus to the bulb has a tortuous course. The blood stream is driven from one wall of the bulb to the other, and thus forms vortices. There is stasis of the blood in the bulb during deep ex-spiration. The widening of the blood stream in the bulb causes a slowing of the current.

The dome of the bulb forms a dead space, that is, a quiet harbor, because none of the tributaries opens into the dome. As a result of the multiplication of the bacteria in the bulb, a thrombus is formed.

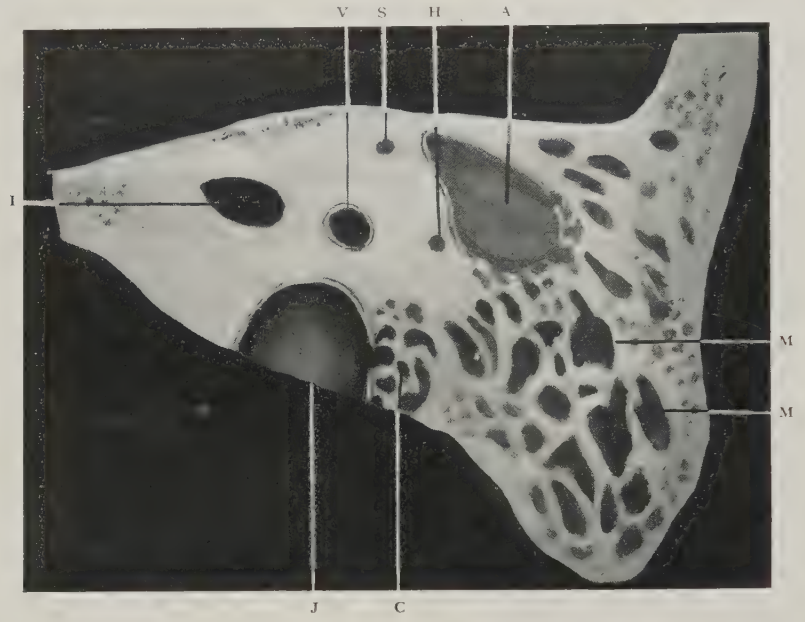

FIG. 78. VERTICAL SECTION OF TEMPORAL BONE, SHOWING CELLS IN CLOSE RELATION TO JUGULAR BULB.

A, antrum; C, cells which lie below posterior wall of external auditory canal, in close relation to jugular bulb; H, horizontal semicircular canal; I, internal auditory canal; J, jugular bulb; M, mastoid cells; S, superior semicircular canal; V, vestibule.

The pressure factor is usually wanting in cases of peribulbar abscess, as the jugular fossa is open in a downward direction, and the pus flows freely down along the jugular vein. A peribulbar abscess may be the cause of the bulb thrombosis, or the result of the thrombosis.

When there is a thrombosis involving both the sigmoid sinus and the bulb, it is often impossible to tell, from macroscopic examination, whether the thrombus was primary in the sinus and extended into the bulb, or vice versa. On microscopic examination, it is usually possible to tell which portion of the thrombus is the oldest.

Of 37 cases of sinus thrombosis autopsied by Grunert, the bulb was free of thrombus in only 13 cases. Of the remaining 24 cases, the thrombus was limited to the bulb in 3 cases.

In 11 cases of sinus thrombosis, Leutert found primary bulb thrombosis 3 times.

Jansen found 2 cases of primary thrombosis in 12 cases of sinus thrombosis.

Secondary thrombosis of the jugular bulb may be due to extension of a thrombus from the sigmoid sinus, the inferior petrosal sinus, the carotid sinus, or the internal jugular vein. When the extension is from the internal jugular vein, the thrombus is usually secondary to an infection in the deep tissues of the neck, most commonly in the lymph glands surrounding the jugular vein. The sequence in these cases is cervical adenitis, perijugulitis, jugulitis, thrombosis of the jugular vein and thrombosis of the bulb.

Primary thrombosis of the superior longitudinal sinus is not very common. When it does occur, it is likely to be of the non-infective or marantic type. Infective thrombosis of the superior longitudinal sinus occasionally occurs with erysipelas of the scalp or suppurative conditions of the scalp. In these cases the infection reaches the superior longitudinal sinus through the emissary vein of Santorini. In osteomyelitis of the bones of the skull, the superior longitudinal sinus may become infected either by direct contact with the involved bone, or through the diploic veins of the skull. In suppurative disease of the nose, especially in disease of the frontal sinus, the superior longitudinal sinus may become involved through a communicating vein which passes through the foramen cecum and reaches the anterior end of the superior longitudinal sinus.

Primary thrombosis of the inferior longitudinal sinus or the straight sinus is exceedingly rare. When it does occur, it is usually marantic.

Isolated thrombosis of the superior petrosal sinus occurs occasionally. It is usually the result of extension from the middle ear and antrum, through the veins which pierce the tegmen tympani et antri. These veins pass through the petrosquamous fissure, and join the veins of the middle-ear mucosa to the superior petrosal sinus. Isolated thrombosis of the superior petrosal sinus may also occur with necrosis of the petrous pyramid.

A thrombus which starts in the superior petrosal sinus may extend backward into the sigmoid sinus, or forward into the cavernous sinus.

Primary thrombosis of the inferior petrosal sinus is somewhat more common than in the superior petrosal. Hessler reported 5 cases of isolated thrombosis of the inferior petrosal sinus. A primary thrombus in this sinus is due either to an inflammatory process in the labyrinth, or to a necrosis of the apex of the petrous pyramid. When there is suppuration in the labyrinth, there may be extension through the veins of the internal ear to the inferior petrosal sinus. The writer reported a case of this kind, where post-mortem examination disclosed necrosis of the labyrinth, phlebitis of the internal auditory vein, thrombosis of the inferior petrosal sinus, and an extension to the cavernous sinus.

Beyer reported a case of isolated thrombosis of the inferior petrosal sinus at the junction of the latter with the jugular bulb. He thought that the origin of the thrombosis in this case was a throat infection, as the pharyngeal vein sometimes empties into the jugular bulb.

When there is a primary thrombosis in the inferior petrosal sinus, it may extend from there into the jugular bulb or into the cavernous sinus.

The primary seat of the thrombus may be in the mastoid emissary. Diseased mastoid cells along the course of the emissary can easily infect this vessel. When the emissary is large, it may cause systemic symptoms similar to those occasioned by thrombosis of the lateral sinus. The thrombus

in the emissary may extend into the sigmoid sinus and produce a secondary thrombus in the latter.

Isolated thrombosis of the cavernous sinus is fairly common. It may result from an infective process on the skin of the face, especially about the nose and lips. It may follow infections in and about the tonsil, in the nasal accessory sinuses and in the middle ear.

Infections on the face reach the cavernous sinus through the facial, nasal and angular veins, and the superior ophthalmic veins. Orbital infections may also reach the cavernous sinus through the ophthalmic veins. Disease of the nasal accessory sinuses may reach the cavernous sinus through the orbital veins. The bulk of the venous return from the nasal mucosa and accessory sinuses is carried to the pterygoid plexus by the sphenopalatine vein. Infection in the accessory sinuses may also travel along the perforating venules to the venous system of the orbit, and from the orbital veins to the cavernous sinus.

Disease of the sphenoidal sinus may cause a cavernous sinus thrombosis by direct extension from the sinus to the vessel wall, as the two structures lie in contact with each other.

Although the infection usually travels through the veins, it may also travel along the lymph channels which accompany the arteries and nerves. Several cases have been reported where the infection travelled along the optic nerve from the orbit to the subdural space, with secondary infection of the cavernous sinus. The olfactory nerves may also serve as pathways for the infection.

Infections in the region of the tonsil travel along the pharyngeal vein to the pterygoid plexus. The pterygoid plexus communicates with the cavernous sinus directly through veins which pass through foramina in the sphenoid bone, and indirectly through the infraorbital vein.

Infection in the middle ear may cause a necrosis of the petrous pyramid. The tip of the petrous pyramid is in contact with the posterior extremity of the cavernous sinus. This may result in primary thrombosis of the cavernous sinus.

FIG. 79. THROMBOSIS OF CAROTID SINUS.
C, internal carotid artery in bony canal; CS, thrombosed carotid sinus.

Middle-ear disease may also cause thrombosis of the cavernous sinus by way of the carotid sinus (Fig. 79). The carotid sinus communicates with the middle-ear mucosa. The carotid sinus may be the primary site of the thrombosis, which extends into the cavernous sinus. The carotid sinus may not contain a thrombus, but may serve as a pathway for the infection from the middle ear to the cavernous sinus, the infection traveling along the wall of the carotid sinus.

Thrombosis of the cavernous sinus may be secondary to thrombosis in the sigmoid sinus or jugular bulb. This extension may occur in one of three ways:

1. By way of the superior or inferior petrosal sinuses, or both.

2. The cavernous sinus may become infected by the retrograde transport of particles of thrombotic material from the sigmoid sinus or bulb.

3. The extension from the bulb to the cavernous sinus may occur by way of the carotid sinus.

Cavernous sinus thrombosis is frequently secondary to meningitis. Meningitis is often accompanied by a thrombophlebitis of the pial veins. As some of the pial veins empty into the cavernous sinus, thrombosis of the latter may readily occur.

Primary thrombosis of the carotid sinus may occur as the result of extension from an infected middle ear, as some of the middle-ear veins drain into the carotid sinus. The carotid sinus may also become involved by direct contact, when there is necrosis of the petrous pyramid. It may become involved secondarily, by extension from thrombosis in the jugular bulb or in the cavernous sinus.

Kramm and Passow have described a number of cases of obliteration of the sigmoid sinus due to compression. Kramm reported 2 cases and Passow reported 6 cases.

Passow found the following conditions present in all of his cases:

1. The bony sinus plate was broken through by disease.

2. There was a perisinous abscess.

3. The outer wall of the sigmoid sinus was diseased.

4. There was no blood in the sigmoid sinus.

5. The outer and inner sinus walls lay in contact with each other, but they were adherent only for a short distance, proximally and distally. In the middle portion they were not adherent. In this area, the sinus was empty.

6. The cerebral wall of the sinus was normal in the non-adherent portion.

7. There were no chills.

In explaining these cases, Passow said: "Due to pressure of the perisinous abscess and thickening of the sinus wall, the outer sinus wall comes to lie against the inner wall. The intima of the outer wall becomes diseased, not only in the area of compression, but also to both sides of it. This results in the formation of a thrombus at both sides of the compressed area. This in turn causes an involvement of the intima of the cerebral wall of the sinus. In this way there results a closure of the sinus above and below. In the compressed area there is no thrombus, because there is no blood there."

If the perisinous abscess is evacuated soon after the approximation of the two walls of the sinus, the circulation may be re-established. Passow saw this happen in the course of an operation.

The organism which is responsible for most of the cases of otogenic general sepsis is the streptococcus hemolyticus. This is also true of general sepsis caused by conditions other than ear infections. Simmons found streptococci in 63 per cent of 1200 cases of general sepsis due to all causes. Lenhartz found 185 streptococcus infections and 51 staphylococcus infections in general surgical sepsis. Streptococci were found in 14 (40 per cent) of 35 cases of otogenic general sepsis observed by Mygind. Staphylococci were found in 10 cases, but only in 3 of these were they present in pure culture. Colon bacilli were found in 7 cases. Pneumococci were found in 1 case. Bacteria of uncertain character were found in 7 cases.

Kobrak found anerobic bacteria in several cases. He believes that bacteria, which in chronic suppurating ears may be only saprophytic and inoffensive, may, when they reach the intracranial cavity, become markedly pathogenic, due to the anerobic conditions in this location.

Marum reported a case due to the Friedlaender pneumobacillus. In 34 cases of otogenic general sepsis examined by Libman, with positive

findings in the blood, streptococci were found in 30 (90 per cent), staphylococcus in 3 (8.8 per cent), and bacillus proteus in 1 case.

Of 23 cases examined by German, 10 were due to streptococci, 6 to staphylococci, 5 to pneumococci, 1 to bacillus proteus, and 1 to mixed infection.

Stenger, as the result of experimental production of sinus thrombosis in dogs, came to the conclusion that bacteria do not cause a thrombus by lodging on the inner surface of the sinus wall; but that the thrombus is caused by changes in the blood, which are indirectly caused by the bacterial invasion. He found that thrombi are formed before the bacteria enter the blood stream, and that infection of the thrombus is secondary.

Talke came to similar conclusions from his experiments. He produced thrombi in the veins and arteries of dogs, cats and guinea-pigs. He found that most of the bacteria were present in the perivascular connective tissue, and that they progress in lesser numbers through the various layers of the vessel walls. In only a few of the cases were they present in the intima. They were present only in the lymph vessels and interlamellar spaces of the vessel walls, and not in the vasa vasorum. Of the 33 vessels experimented upon, bacteria were present in the thrombus in 15 cases; in 18 vessels the thrombus was sterile. The thrombi which contained bacteria had them mostly in the portion nearest the vessel wall. The youngest thrombi contained no bacteria. Of the one-day-old thrombi, 9 were sterile and 3 contained bacteria; of the two-day-old thrombi, 7 were sterile and 3 contained bacteria; of the three-day-old thrombi, 1 was sterile and 10 contained bacteria. He found that the inflammation in the vessel wall travelled toward the intima faster than the bacteria.

Mygind made cultures from thrombi which were removed at operation in 60 cases; in 20 of these the thrombus was not infected.

Haymann made some very interesting observations in the course of his experimental production of sinus thrombosis in dogs and monkeys.

He found that a thrombus can develop within a few hours after infection of the outer surface of the sinus wall. It begins with the formation of a parietal thrombus (Figs. 80 and 81). The growth may occur by the gradual

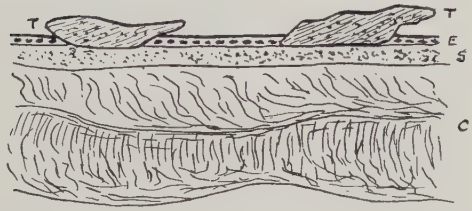

FIG. 80. TWO PARIETAL THROMBI LYING ON INNER WALL OF SINUS. NO ENDOTHELIUM UNDER THROMBI. INTACT ENDOTHELIUM BETWEEN AREAS COVERED BY THROMBI. SINUS WALL THICKENED. (LOW MAGNIFICATION.) (*After Streit.*)

C, connective tissue layer of sinus wall; E, endothelium; S, subendothelial tissue; T, thrombi.

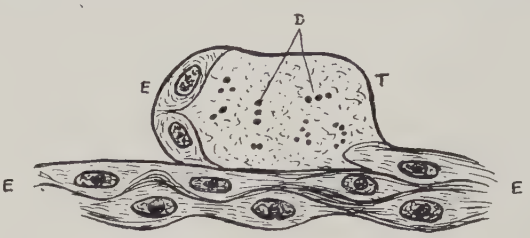

FIG. 81. BLOOD PLATELET THROMBUS LYING ON ENDOTHELIAL LAYER OF INTIMA. (HIGH MAGNIFICATION.) (*After Streit.*)

B, bacteria; E, endothelium; T, thrombus.

apposition of new layers of thrombus, until the thrombus is obturating. It may remain parietal. Occasionally the entire blood column may clot at once. When this occurs, the case usually runs a malignant course.

He found that there must be a continuous infection of the sinus wall, for a considerable period of time, in order to produce a thrombus. Narrowing of the sinus by compression of a sterile plug does not produce thrombosis. Infection must be added to the compression. Jakowski and Haymann found that if the sinus is narrowed by the compression of a sterile plug, and bacteria are introduced into the circulation at some other point, as, for instance, through the femoral vein, a thrombus is formed at the point of compression.

The virulence and type of infection are important both in experimental and in otogenic sinus thrombosis. In the latter, certain types of bacteria are

so commonly present, that they must be allowed a particular disposition to the formation of this complication.

Otogenic general sepsis is produced in most, but not in all cases, by sinus thrombosis. Haymann found, in two of the dogs, and in one of the monkeys, that bacteria passed through the sinus wall directly into the blood stream. This occurs frequently in cases of gonorrhoeal general infection.

The sinus is not the only possible pathway for the infection to take in order to reach the general circulation from the ear; other veins may act as intermediaries. Clinically, it is impossible to differentiate otogenic general infection with sinus thrombosis, from otogenic general infection without sinus thrombosis. In the latter class of cases, incision of the sinus with tamponade may produce a thrombosis.

Although the thrombus is usually infected after it is formed, Haymann found that occasionally it is infected as soon as it is formed.

He found that parietal thrombi may undergo organization as such. Parietal thrombi are not always infectious.

He found that the different parts of obturating thrombi may be of different appearance. Some parts may be rich in fibrin, while other parts contain mostly red cells, white cells or platelets. Various stages of organization may be present in different parts. The individual layers of the same thrombus may show differences in age.

When a thrombus breaks down, the changes are usually most marked in the center. Most of the thrombi in the experiments contained bacteria. The location of the bacteria depends upon the mode of development of the thrombus. The older portions which are partly organized are usually bacteria-free, while the more recent portions contain bacteria. In many cases it can be seen how bacteria are deposited with or on the newly deposited layer of the thrombus. Occasionally, the older portions of the thrombus contain bacteria, while the recent portions are sterile. Organization of the thrombus is not

interfered with by the presence of a moderate number of bacteria. Haymann found that there is a marked tendency for all thrombi to organize. Organization does not necessarily mean healing. Organization is found in thrombi which show definite breaking down in some parts. This partial organization cannot prevent general sepsis. It may occur very early. In 14 out of 19 animals experimented upon, there was some organization in the thrombus. It was found in one case as early as three days after infection. Organization may be complete in fourteen to twenty-one days.

Haymann found that the wall of the sinus is usually infiltrated with granulations, and thickened. It may contain areas of purulent breaking down, or it may be eroded. There may be bloody infiltration of the wall, or edema. The wall may be completely destroyed. Where the thrombus is adherent to the wall, the endothelium is usually destroyed. It may be thickened. The thrombus may produce changes in the sinus wall secondarily.

He found that an aseptic incision of the sinus, even when followed by tamponade, produces no ill results. It produces only a very small deposit on the intima. When there are bacteria in the circulation, aseptic incision of the sinus with tamponade causes a thrombus formation, with breaking down of the outer wall of the sinus.

The macroscopic appearance of the thrombus gives no idea as to its infectivity, unless it is broken down. The macroscopic appearance of the sinus wall may give no idea of the contents of the sinus, unless the wall is broken down.

Haymann found that ligation of the jugular vein produced no thrombosis in the vein, nor did it increase the thrombosis in the sinus.

Bacteria enter the circulation from the thrombophlebitic focus either enclosed in a particle of thrombotic material, or else free. The mode of entrance of bacteria into the circulation may occur in one of four ways:

1. When the thrombus is obturating, bacteria are carried through veins which open into the sinus near the thrombosed area. A retrograde circulation is set up in these veins as the result of the blocking of the sinus.

2. When the thrombus is of the mural type, bacteria and particles of thrombus may be washed from the free surface of the thrombus by the blood stream, as it flows past the involved area.

3. When the thrombus is obturating, it may break down in part, and the circulation may become partly restored.

4. Bacteria which are lodged in the sinus wall may pass into the perivascular spaces and the vasa vasorum, and into the dilated vessels in the connective tissue about the sinus, and they may thus be carried into the general circulation.

The secondary results of thrombophlebitis of the venous sinuses of the skull may be divided into local results and distant results.

In thrombophlebitis of the sigmoid sinus, a perisinous abscess may result from the infective process in the sinus. The pus from this abscess may extend along the mastoid emissary, through the emissary canal, and give rise to an abscess at the back of the neck, about the point of exit of the emissary. A thrombus in the emissary vein may break down and give rise to a similar result. The pus from a perisinous abscess may flow alongside the posterior condyloid emissary through the posterior condyloid canal. It makes its exit at the under surface of the occiput, and gives rise to an abscess under the deep tissues of the neck, in the upper third of the posterior cervical triangle.

Thrombosis of the jugular bulb may result in a gravity abscess in the neck. It may cause an intravenous abscess, or an extravenous abscess. An abscess within the bulb descends through the internal jugular vein. If the vein is not incised, the disease will infiltrate the perijugular tissues and may perforate the wall of the vein, and form a perivenous abscess. The lymph

glands about the jugular vein also become inflamed and may break down. An extravenous abscess or peribulbar abscess results from infection of the peribulbar connective tissue, from contact infection from the diseased bulb wall, or through a fistula in the wall of the bulb. The collection of pus may be on the medial or on the lateral side of the bulb. It may extend upward to form a perisinous abscess, or downward along the internal jugular vein. It may extend down until it perforates the pleura or the mediastinum.

A peribulbar abscess may extend horizontally along the base of the skull and perforate into the pharynx.

The inflammatory process may extend from the bulb through the condyloid foramina, either through thrombosed veins in the foramina, or outside of the condyloid veins. When the infection passes through the anterior condyloid canal, it extends along the sheath of the hypoglossal nerve, and may cause an inflammation of the nerve. Infection passing through either of the condyloid canals may cause an abscess under the deep cervical fascia, in the upper third of the posterior cervical triangle. It may result in periarticular or articular involvement of the joint between the occiput and atlas. There may be caries of the condyle. There may also be caries of the posterior part of the upper cervical vertebrae. The membrana obturatoria posterior between the occiput and the atlas may be perforated, and the pus may enter the spinal canal.

When the thrombophlebitic process in the sigmoid sinus extends to the visceral wall, the condition becomes a pachymeningitis interna, for the visceral wall of the sinus constitutes part of the dural covering of the cerebellum. When the entire thickness of the visceral wall becomes involved, and the disease reaches the inner surface of this wall, an exudate may collect on the inner surface and form a subdural abscess. Sometimes the dura becomes adherent to the surface of the cerebellum at this point, and there occurs either a cerebellar abscess or a meningitis.

Numerous small veins pass from the sinus to the brain. The veins are surrounded by perivascular lymph spaces. The infection may travel from the sinus along or within these vessels to the brain.

Brain abscess or meningitis may occur as the result of sinus thrombosis in one of three ways:

1. Disease of the visceral sinus wall may extend by continuity to the meninges and brain substance. The extension may be through the small veins or lymph vessels, or through the connective tissue.

2. A piece of thrombus or loose bacteria may be carried from the sinus into the general circulation, and after passing through the lung, lodge in one of the small vessels in the brain or meninges.

3. The most common method is by retrograde transport of infectious material or by retrograde extension of the thrombus from the sinus into the pial or cerebral veins.

Heilbronn reported 3 cases of focal softening of the brain due to retrograde thrombosis of the cerebral veins. In one of these cases there were general cerebral symptoms. Post-mortem examination showed retrograde extension of the thrombus from the lateral sinus to the vena magna Galeni and the small basal cerebral veins, with spots of hemorrhagic softening in both optic thalami. In another case, with disturbances of speech, multiple small lesions were found in the medulla. In a third case, there was aphasia and agraphia lasting for a day. These symptoms were due to an extension of the thrombus from the lateral sinus to the large pial veins which surround the temporosphenoidal lobe and drain it. This caused an edema of the temporosphenoidal lobe, which disappeared as soon as a collateral circulation was established.

In 77 cases of sinus thrombosis treated at the University clinic in Budapest, German found intracranial complications in 50 (65 per cent). Of these, perisinous abscess was present in 27 cases; subdural abscess alone, in

no case; meningitis alone, in no case; brain abscess alone, in 3 cases; perisinous abscess with subdural abscess in 1 case; perisinous abscess with subdural abscess and cerebellar abscess in 1 case; perisinous abscess with brain abscess in 7 cases; perisinous abscess with meningitis in 4 cases; perisinous abscess with meningitis and brain abscess in 2 cases; subdural abscess with meningitis in 1 case; and brain abscess with meningitis in 4 cases.

Hinsberg found that of all otitic meningitides, 6.5 per cent were due to sinus thrombosis. Of 77 cases of sinus thrombosis observed by German, 11 had meningitis.

Bacteria in the spinal fluid, with increase in pressure and increase in cells and albumin, in cases of sinus thrombosis, indicate meningitis. The presence of bacteria without inflammatory changes in the spinal fluid, in cases of sinus thrombosis, has no significance. Voss found that in uncomplicated sinus thrombosis, bacteria were present in the spinal fluid in one quarter of the cases.

In cavernous sinus thrombosis, there may be pus foci in the cellular tissue of the orbit, especially along the ophthalmic vein. There may be edema of the orbital tissue without abscess formation, due to interference with the return circulation from the orbit. As a result of extension of the thrombotic process from the cavernous sinus to the pterygoid plexus, there may be an edema of the pharynx. Through extension of the process to the facial vein, there may be edema of the face. Jansen reported a case of cavernous sinus thrombosis, in which the pus from the cavernous sinus broke through into the sphenoid sinus. In thrombosis of the superior or inferior longitudinal sinuses, retrograde extension of thrombi into the meningeal or cerebral veins may cause areas of necrosis or hemorrhagic softening or edema of the brain or meninges. Whether these secondary changes in the brain are suppurative or not, depends upon whether the thrombus in the sinus is marantic or infective.

Metastases. The distant secondary results of thrombophlebitis of the venous sinuses of the skull are the metastases.

Embolic pieces of infected thrombotic material or free bacterial emboli may be carried into any part of the tissues within the skull by retrograde transport. They may also be carried to the superficial tissues of the head and neck in the same way, after passing through one of the emissary veins. In order to reach any other part of the body, the emboli must first pass through the capillaries of the lung. If the embolus is too large to pass through the pulmonary capillaries, it will be held up in the lung and produce a pulmonary metastasis. If the embolus consists of a single bacterium or a small group of bacteria it may pass through the pulmonary capillaries and into the peripheral circulation. It may then lodge in any part of the body and give rise to a metastasis in any structure. The observations of the older otologists seemed to show that pulmonary metastases were more common with chronic middle-ear suppuration, and metastases in the peripheral circulation were more common with acute middle-ear suppuration. They ascribed this to the fact that with chronic otitis the emboli were more likely to consist of infected pieces of thrombotic material, whereas, in acute otitis, the emboli were likely to consist of free bacteria. More recent statistics show, however, that both types of metastases occur with equal frequency in both acute and chronic otitis. Metastases occur after the thrombophlebitis has existed for some time. They never occur in the first few days of the thrombophlebitic process. The early stages of the thrombophlebitic process consist in an attempt on the part of the sinus to hold back and localize the invading infection, by the formation of a thrombus. If it does not succeed in this, and bacteria enter the general circulation in the first few days of the disease without the formation of a thrombus, the result is not the occurrence of metastases, but an overwhelming of the system by the poisons elaborated by the bacteria in the circulation, and an early death with acute septic

symptoms. When the infection is comparatively mild, and the sinus succeeds in building up a thrombus, the infection may remain localized in this area long enough to allow a removal of the original infective process; in other words, the performance of a mastoidectomy. If a mastoidectomy is done, and the sinus uncovered, and perhaps a perisinous abscess evacuated, the thrombus may organize and heal, and the patient get well without the occurrence of metastases.

In the cases where the infection is of medium severity, and after the thrombus has been in existence for some time, usually from a few days to a few weeks, and the infection from the original focus in the ear and mastoid continues to pass into the thrombus, the thrombus breaks down and throws bacteria into the general circulation. In this way, metastases occur. In other words, the very severe and the very mild infections have no metastases. The moderately severe infections have metastases if the infective process in the original focus and in the sinus is allowed to continue.

Metastases may occur in the skin, in the subcutaneous tissues, in and about the joints, or in any of the internal organs. The organs which are most commonly involved are the lungs and the brain, but any of the other organs may be the seat of the metastasis: kidneys, liver, spleen, heart or gastro-intestinal tract.

Every metastatic focus does not go on to abscess formation. There is a condition called dermatomyositis, which is characterized by scattered areas of painful swellings in the skin and muscles in different parts of the body. These swellings disappear in a few days, without breaking down. Histological examination of these lesions by A. Fraenkel showed that the area of involved muscle was edematous with hemorrhages, the perimysium was edematous; and the area was covered by edematous erysipeloid-colored skin. It is believed by some otologists that these are not metastatic areas, but are due to toxins liberated by the bacteria in the thrombophlebitic area.

It is more likely, however, that they are metastases in which the bacteria are so few, or so non-virulent, that no breaking down of tissue occurs. Similar metastatic foci occur frequently in the periarticular tissues. The joint becomes painful and swollen, and gets well in a few days. The metastatic foci in the joints, which go on to suppuration, may start either in the periarticular tissues, in the bone near the articular surfaces of the joint or in the joint itself. When it starts around the joint, it may later break into the joint.

Metastases in the lung start as infarcts. They occur as pyramidal shaped areas of necrosis, which often involve the pleura. If the infectivity of the embolus is very slight, the involved area gradually cicatrizes. If the embolus contains many bacteria the lung tissue breaks down and forms an abscess. Metastatic abscesses in the lung are usually multiple. When the pleura is involved empyema often occurs. The lung abscesses may rupture into a bronchus, or into the pleural cavity. Erosion of a large vessel does not occur in this type of abscess as frequently as in tubercular abscesses.

Metastases in the brain may cause areas of focal softening when the embolus is not very infectious. Temporary brain symptoms due to edema of the brain may occur, but they disappear when the embolus is dislodged from the vessel in which it was caught, or after collateral circulation is established. When the embolus is very infectious, subdural abscess, brain abscess, or meningitis results.

The inflammatory process which extends from the sinus to the meninges sometimes gives rise to an involvement of one or more of the cranial nerves. This is especially true of the third, fourth, fifth and sixth nerves. The nerves may become inflamed as the result of a direct extension of the inflammatory process from the meninges. In thrombosis of the cavernous sinus, the inflammatory process spreads directly from the thrombus and the inflamed sinus wall to the nerves which pass through the cavernous sinus. Inflammation of the ninth, tenth and eleventh nerves sometimes occurs as the result of

extension of the inflammatory process from the jugular bulb and the internal jugular vein to these nerves as they pass through the jugular foramen. Inflammation of the optic nerve occurs quite frequently with sinus thrombosis. It may be due either to a meningitis or to an obstruction to the circulation.

Metastases in the kidneys may result in infarcts or abscesses in these organs. Metastases in any of the abdominal organs may result in the formation of abscesses which sometimes perforate into the peritoneal cavity, giving rise to a peritonitis.

Chapter IV

SYMPTOMS AND DIAGNOSIS

Chapter IV

SYMPTOMS AND DIAGNOSIS

THE clinical picture of otogenic general sepsis is a varied one. The typical picture as described in textbooks is far from typical because it occurs in only a small proportion of the cases. The so-called atypical cases are more numerous than the typical ones. With the development of the newer diagnostic methods, we are enabled to bring to light many cases of otogenic general sepsis which were formerly overlooked, because they presented so few symptoms.

A thrombophlebitis of one of the cerebral sinuses may run its course with no symptoms whatever and may be discovered accidentally during the performance of a mastoid operation. Otogenic general sepsis sometimes manifests itself as a very acute virulent sepsis, with continuous high fever, disturbances of the sensorium, rapid feeble pulse, and death in a few days.

Most of the cases lie between these extremes. They present the clinical picture of septicopyemia, with sharp rises and falls in the temperature, chills and sweats, and the occurrence of metastases. The nearer the picture approaches the pyemic type, the easier is the diagnosis.

The symptoms and signs that may be present in thrombophlebitis of the cerebral sinuses can be grouped into general symptoms and local symptoms. The general symptoms are those of sepsis and pyemia.

The local symptoms may be divided into:

1. The external signs about the head and neck caused by obstruction to the circulation and by extension of the inflammation from the sinus to the surrounding tissues.

2. The symptoms due to interference with the cerebral circulation and to extension of the inflammatory process from the sinus to the adjacent brain, membranes and nerves.

3. The changes which are found in the sinus wall, and within the sinus, when the latter is exposed during a mastoid operation.

General Symptoms

Temperature. The most important of the general symptoms is fever. The fever of otogenic general sepsis is frequently of the pyemic type. There are sharp rises and falls in the temperature every day. The drop in the temperature is to normal or subnormal, and the rise is to 105° F. or 106° F. The duration of the individual rise and fall from low to low temperature varies between six and thirty-six hours. It averages twenty-four hours. When there is more than one rise and fall in twenty-four hours, the diagnosis of sinus thrombosis is fairly certain, as there are very few other conditions in which the febrile curve has this character. The length of time from the lowest to the highest temperature may be one to three hours. The temperature may remain at the peak only one or two hours, and the drop in the temperature may be as sudden as the rise. There may be one or two secondary peaks on both the up and the down curves. In other words, the rise and the fall may not be represented by straight lines, but by notched lines.

Takabatake analyzed the temperature curve in 51 cases of typical otogenic pyemia, and noted the time of day at which the peak of the temperature occurred.

It occurred from twelve o'clock midnight to four A.M., 10 times; from six A.M. to ten A.M., 11 times; from midday to four P.M., 12 times; from six P.M. to ten P.M., 18 times.

He found that the lowest temperature was below 99.5° F. in 62 per cent of the cases, and above 99.5° F. in 38 per cent of the cases.

In the septic cases, there is a continuously high temperature, like that in pneumonia. In some cases, the temperature does not go above 102° F. or

103° F. every day. It may go down to normal or subnormal every day, or it may drop only 1 or 2 degrees daily.

In some cases the temperature is normal at all times. This is especially likely to occur in the cases where the abscess within the sinus has ruptured through the outer sinus wall, and the infective material in the sinus drains into the mastoid cavity. It also occurs in cases where the thrombus is sterile and has undergone partial or complete organization.

Chills. Chills frequently occur with each sharp rise in the temperature. The chill usually starts shortly after the beginning of the rise in temperature, and it may last until the temperature has reached its maximum. It sometimes lasts from half an hour to an hour. It may be very violent, the patient becoming very pale, with blue lips, and the entire body shake violently. The application of heat to the body has no effect upon the chill. In many cases, the patient only complains of slight chilly sensations, and a feeling as of goose-flesh. Chills occur in less than half of the cases of otogenic general sepsis. In some cases chills only occur in the early stages of the disease, and they may be so slight that they are overlooked by the patient and his attendants. In children chills are uncommon but they are frequently replaced by convulsions.

The chills are usually followed by sweats. The sweating coincides with the drop in temperature.

Mechanism of Chills. The periodicity of the rises in temperature is due to the fact that bacteria are liberated from the thrombus and flood the circulation at periodic intervals. When the circulation is flooded by bacteria, there is a sharp rise of temperature with a chill. As the bacteria are destroyed by the bactericidal action of the blood, the temperature falls.

A rise in temperature is caused by the breaking down of the protoplasm of body cells and of bacteria, and the consequent liberation of foreign substances in the circulation. These substances attack the heat-regulating

centers, which are located in the gray matter ventral to the optic thalami. These centers are related to metabolism, to innervation of the skin and respiration, and to the loss of water from the skin and lungs.

Heat production results from the burning up of the body-tissues, especially in the muscles, liver, adrenals, thyroid and hypophysis. During fever, the same substances are burned up as in the healthy body. During fever, excretion of water by the skin is increased. This causes some loss of heat by evaporation of the water. When the drop in temperature occurs in a very short time, sweating is greatest. There is also loss of body-heat by evaporation of water in the lungs, and by conduction and radiation from the skin. In this way the temperature of the body is reduced.

When the increase in heat production is greater than the increase in heat dissipation, fever results. When the increase in heat dissipation overtakes the increase in heat production, the body temperature returns to normal.

When very large quantities of fever-producing materials are introduced into the body, they may produce a paralysis of the heat-regulating center. This causes a sudden fall in the temperature, and death may result. This occurs in the collapse of acute infectious diseases, and also in anaphylactic shock.

A chill is the expression of an intensive effect upon the vasomotor nerves of the skin vessels, by the poison which enters the circulation. A spasm of the vasoconstrictors of the skin produces a feeling of cold and causes the chill.

Pulse. The pulse is usually rapid in otogenic general sepsis. It is ordinarily of good quality. In the cases of very severe sepsis, it may be weak and soft. The pulse rate usually follows the temperature, increasing in rapidity when the temperature rises, and decreasing in rapidity when the temperature falls. Takabatake compared the pulse rate and temperature in a large

number of cases of otogenic general sepsis, and found the following relationship between the two:

When the temperature was between 97° F. and 98.6° F., the pulse rate

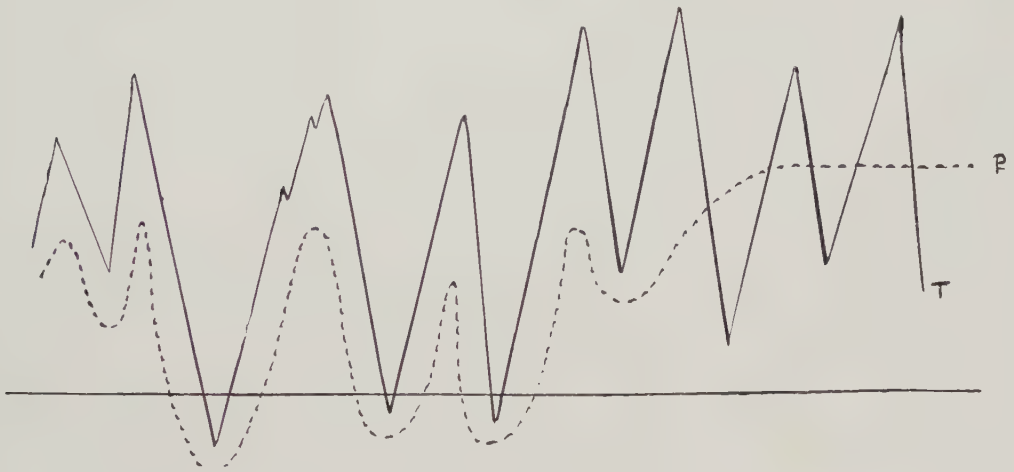

FIG. 82. GRAPHIC TEMPERATURE AND PULSE RATE CHART SHOWING DEATH-CROSS.

P, pulse rate; T, temperature.

averaged 89; between 98.6° F., and 100° F., the pulse rate averaged 91; between 100° F., and 102° F., the pulse rate averaged 113; between 102° F. and 104° F., the pulse rate averaged 126; over 104° F., the pulse rate averaged 134.

In children the pulse rate is usually higher than in adults.

When the pulse rate remains persistently high and does not come down with the drop in the temperature, the prognosis is bad. This persistence in the rapidity of the pulse is usually due to myocardial degeneration or endocarditis. A persistently high pulse rate with sharp rises and falls in the temperature, produces a cross in the graphic temperature and pulse rate chart. This cross has been termed a "Todeskreuz" or "death-cross" by Mygind (Fig. 82). A "death-cross" may also occur when a sinus

thrombosis is complicated by some intracranial condition which causes a slow pulse.

Mental Condition. The toxemia of the disease frequently causes a mental stimulation and a feeling of euphoria. Patients with very high temperature frequently feel almost normal. In fact, this condition is so characteristic, that when there is a very high temperature with middle-ear suppuration, and the patient feels very well, one should strongly suspect the presence of a sinus thrombosis. In some cases, periods of euphoria are followed by periods of depression.

There is usually loss of appetite with a coated tongue. There may be fetor ex ore; nausea and vomiting; constipation or diarrhea. Herpes occasionally occurs about the mouth. The patients have a pale pasty look, due to the secondary anemia which is always present. During the chill, the lips may become blue. At the height of the temperature, the face is usually flushed. As a rule there is considerable loss of weight.

Blood Picture. The anemia is sometimes very marked in otogenic general sepsis. The hemoglobin may drop to 35 or 40 per cent. The anemia is the result of the destruction of the red blood cells by the bacteria in the circulation. When the anemia is progressive, the prognosis is bad. When the blood becomes sterile as the result of operative intervention, the progress of the anemia is stopped. A progressive anemia after operation means that the sepsis has not been controlled.

There is an increase in the total number of leucocytes in otogenic general sepsis; the increase may be up to 18,000 or 20,000. There is an increase in the polymorphonuclear neutrophiles, usually above 80 per cent. There is a loss of the eosinophiles, and a diminution of the lymphocytes to 10 or 15 per cent.

The normal differential leucocyte count is as follows:

Basophiles 1 per cent.

Eosinophiles 2 to 4 per cent.

Neutrophiles 67 per cent { unsegmented 3 to 5 per cent. / segmented 62 to 64 per cent.

Lymphocytes 21 to 35 per cent.

Mononuclears 4 to 8 per cent.

The eosinophiles diminish in septic infections; the more severe the infection, the more completely do they disappear. The neutrophiles increase and the lymphocytes diminish in acute purulent inflammation. The lymphocytes increase in chronic inflammations. If the total leucocytes do not increase, with an increase in the polynuclear neutrophiles, it indicates a loss in the resisting power of the patient.

Bacteriemia is probably present at some time in every case of otogenic general sepsis. It is not always possible to demonstrate it. When there is a bacteriemia, the blood usually contains only one type of bacteria. There may be several types of bacteria in the middle ear and mastoid, and even in the thrombus. But only one type is "blood pathogenic," while the others probably have a saprophytic character, and if the latter get into the circulation, they are quickly destroyed.

German took blood cultures in 42 cases with pyemic symptoms. There was no demonstrable thrombus in the sinus in 12 of these cases. There were metastases in 15 of the cases. Positive blood cultures were found in 23 of the 42 cases, and negative cultures in 19 cases. Of the positive cultures, 10 were streptococci; 6 were staphylococci; 5 were pneumococci; 1 was bacillus proteus and 1 was a mixed infection. Of the 15 cases with metastases, 7 had bacteriemia. Of 27 cases without metastases, 16 had bacteriemia.

In 40 cases of sinus thrombosis observed by Atkins, positive blood cultures were found in 51.5 per cent. Streptococci were found in all except 4 cases; staphylococci were found in these.

Libman reported the results of the blood examination in 43 cases of sinus thrombosis. Positive blood cultures were obtained in 34 cases. In these 34 cases, streptococci were found in 30 (90 per cent); streptococcus mucosus in 3 (8.8 per cent); and bacillus proteus in 1 case.

The technique employed by Libman in examining the blood is as follows: "Ten to 25 c.c. of blood are withdrawn from a vein (usually an arm vein) and incubated in agar, glucose-agar, serum-agar, glucose-serum-agar, and in bouillon and glucose-bouillon, with and without the addition of ascitic serum. Plates and flasks are incubated for five days, subinoculations being made daily from the flasks (after shaking) in glucose-bouillon and on glucose-serum-agar."

Leutert, who was the first to utilize blood cultures in the examination for bacteriemia in sinus thrombosis, compared the blood aspirated from the lateral sinus with the blood drawn from an arm vein. He believes that if there are more colonies in the sinus blood than in the blood from the arm vein, or if there are bacteria in the sinus blood and none in the blood from the arm vein, we are justified in making a diagnosis of sinus thrombosis. The lesser number of colonies in the blood from the arm vein is due to the fact that some of the bacteria are destroyed by the bactericidal action of the blood, shortly after they enter the general circulation. If the same number of colonies is present in the sinus blood as in the blood from an arm vein, Leutert thinks that the diagnosis of sinus thrombosis is likely, but not positive.

This method of examination, however, is open to the criticism that the outer surface of the sinus is usually contaminated, even after careful disinfection, and the blood which is drawn from the sinus may be contaminated from this source. Furthermore, even if the surface of the sinus is rendered completely sterile, there may be bacteria in the tissue of the outer sinus wall, which may enter the needle as it passes through.

The results which are obtained when the blood is drawn from an arm vein are much more reliable, as the field can be easily sterilized by painting the skin with iodine. In young children, when the arm vein is too small, the jugular vein may be utilized to obtain blood. Sometimes it is necessary to puncture the superior longitudinal sinus.

Ottenberg described a method of examination, recently, in which the blood was taken from both internal jugular veins. In infants and in persons with arm veins from which it is difficult to secure blood, the internal jugular vein is the easiest vein in the body from which to secure blood. His description of the method is as follows: "A small sharp needle (1½ inch long, 19 gauge) is inserted at about the level of the hyoid bone, at the anterior third of the sternomastoid muscle, and is pushed downward and inward toward the manubrium so as to pierce the sternomastoid obliquely. The internal jugular vein is superficial, being covered only by the anterior part of this muscle. The patient is instructed to hold his breath and press, in order to distend the vessel. Four c.c. of blood are obtained from each jugular."

In a case examined in this way, more colonies were found on the normal side. This may be explained by the fact that when there is an obturating thrombus in the sigmoid sinus and bulb, the upper part of the internal jugular vein forms a cul-de-sac, which has no tributaries. The blood in it is relatively stagnant. The blood current above the thrombus is reversed and finds its way through the torcular and through the petrosals to the opposite internal jugular vein. The opposite internal jugular vein carries off most of the blood from the head, and consequently contains most of the bacteria which are washed away from the thrombus. In the case of a mural thrombus, the larger number of bacteria would be found in the jugular of the affected side. For this reason, the usefulness of this procedure in determining on which side the thrombus lies is limited.

The vein from which the blood is drawn is not as important as the time when it is drawn. The blood culture is most likely to be positive, if the blood is taken during a chill, or immediately after. For it is at this time that the circulation is flooded with bacteria. Repeated blood cultures should be taken if the first one is negative. It not infrequently happens that the first two or three cultures are negative, and a later culture is positive. Repeated cultures are also of value to determine the course and the prognosis of the disease.

Libman found positive blood cultures only in cases of sinus thrombosis and meningitis. Positive cultures may also occur in acute tonsillar infections, acute bacterial endocarditis, pneumonia, typhoid fever, and scarlet fever. The number of bacteria found in a cubic centimeter of blood varies from one to many hundred.

The cultures usually become negative a very short time after operation on the sinus and ligation of the jugular. In a case examined by Libman, the blood was sterile eight hours after operation on the sinus. It is often sterile one to two days after operation.

If bacteria persist in the blood after operation on the sinus and ligation of the jugular, it means either that the thrombus has not been completely removed; or that it has extended after the operation; or that multiplication of bacteria in the blood occurs; or that meningitis has developed; or that there is an acute bacterial endocarditis. Metastases do not usually cause a bacteriemia.

Libman gives the following possible reasons why the blood culture may be negative in cases of sinus thrombosis:

"1. The organisms may be so few in number that none is obtained in the amount of blood used.

"2. Anerobic organisms may be missed by not making use of a proper technique, that is, by not using proper culture media.

"3. The cultures may have been made before the organisms were present in the blood.

"4. The cultures may have been made after there are no more bacteria discharged from the local focus. This may be due to:

a. organization of the clot

b. the thrombus breaks down, but instead of separate bacteria, pieces of thrombus enter the circulation.

"5. The bacteriemia may be intermittent.

"6. The bactericidal power of the blood may be so marked that the bacteria are rapidly killed off.

"7. The ends of the clot may be bacteria-free, even though the central part is infected.

"8. The thrombus may close the vessel in such a way that no bacteria are carried into the circulation.

"9. The thrombus may be bacteria-free."

Kobrak suggests that the bacteriological examination of a case of otogenic general sepsis should cover the following points in order to be complete:

1. Determination of the organisms in the ear-pus.

2. Determination of the bacteria in the sinus contents (blood, pus or thrombus).

3. Blood cultures from an arm vein.

4. Agglutination of the ear-flora by the blood serum of the patient.

5. Determination of the organisms in the metastases.

6. Determination of the behavior of the blood serum of the patient toward the bacteria in the metastases and also toward the bacteria in the blood.

He advises that anerobic blood cultures should be taken in all cases of sepsis due to chronic middle-ear suppuration.

A positive blood culture in the presence of middle-ear suppuration is not absolutely diagnostic of sinus thrombosis, but it constitutes very strong evidence of the presence of this condition. If the other conditions in which bacteriemia might occur can be eliminated, and they frequently can be eliminated, the diagnosis of sinus thrombosis is positive. A negative blood culture is not of much assistance in making a diagnosis, because in many cases of sinus thrombosis there is no demonstrable bacteriemia.

Spinal Fluid. The spinal fluid sometimes contains bacteria in cases of sinus thrombosis. When there are no changes in the spinal fluid except the presence of bacteria, and perhaps some increase in the spinal fluid pressure, we need not assume the presence of a meningitis. However, if in addition to the presence of bacteria, there are an increase in the cell content of the fluid and an increase in the albumin content, the sinus thrombosis is complicated by a meningitis.

Metastases. Metastases should be included among the general symptoms of otogenic general sepsis, as they are the result of the bacteriemia. Metastases occur in less than half of the cases of sinus thrombosis. They may occur in any part of the body. They give the symptoms of abscesses in the organs which are attacked.

The localization of metastases usually remains in the same organ during the entire course of the disease. For example, if the first metastases occur in the muscles and subcutaneous tissues, all of the succeeding metastases in that patient occur in the muscles and subcutaneous tissues. If the first metastases occur in the lung, only lung metastases occur throughout the course of the disease in that patient. If the first metastases occur in the joints, only joint metastases are likely to occur in that patient. However, in exceptional cases, metastases may occur in several different organs in the same patient.

Metastases in the muscles and subcutaneous tissues manifest themselves as painful reddened fluctuating swellings on the limbs, back, abdomen, chest, scalp or face. These metastases sometimes break down, but they frequently resolve without breaking down.

Metastases in the lung usually start with the picture of a pulmonary infarct. There is sharp stabbing pain in the chest, cough, and sometimes bloody expectoration. At first there are no physical signs. Later, râles are heard over the affected area. When a lung abscess forms, there is foul-smelling profuse expectoration, and if the abscess is large enough, the physical signs of a cavity in the lung may be elicited. A roentgen-ray picture usually shows the abscess. The infarct does not always develop into an abscess. If the abscess ruptures into the pleural cavity, signs and symptoms of empyema or pyopneumothorax develop.

Metastases into or around the joints manifest themselves as reddened, painful swellings of the joints, the swellings frequently disappearing in a few days. Aspiration of the joint may disclose a purulent exudate.

Metastases in the brain and meninges give symptoms of brain abscess or meningitis.

Metastases in the gastrointestinal tract cause abdominal pain, nausea and vomiting. If the abscess perforates into the abdominal cavity, symptoms of peritonitis supervene.

Metastases in the kidneys manifest themselves by the usual signs and symptoms of infarcts or abscesses in the kidneys.

Among the more unusual sites for the occurrence of metastases may be mentioned the larynx, eye and heart.

Ballance reported a metastatic abscess in the larynx. Several cases of metastatic panophthalmitis have been described. Voss reported a metastatic abscess in the heart muscle.

Hirsch reported a case of metastatic abscess in the interventricular septum of the heart, involving the bundle of His. This resulted in Stokes-Adams syndrome. There was a very slow pulse, and dissociation between the venous and arterial pulse. The pulse went down to 18, but it was regular and strong. The venous pulse in the neck was not synchronous with the arterial pulse. There were two or three venous pulses to one arterial pulse. This was due to dissociation between the auricles and the ventricles of the heart.

Local Symptoms

External Signs. The local signs and symptoms of sinus thrombosis vary with the sinus which is the seat of the thrombus.

When the sigmoid sinus is the seat of the thrombus, the signs due to obstruction to the circulation on the surface of the skull are very slight or absent altogether. When the thrombus extends to the mastoid emissary, there is edema and tenderness at the point of exit of the emissary. This is along the posterior margin of the mastoid. This is called Griesinger's sign, and is differentiated from the edema caused by uncomplicated mastoiditis by the fact that in sinus thrombosis, the edema and tenderness do not extend anterior to the posterior border of the mastoid. Griesinger's sign is not diagnostic of thrombosis of the emissary, as it may also be present when there is a perisinous abscess, with pus trickling through the emissary canal alongside the emissary vein.

When there is a thrombus in the posterior condyloid emissary, or when pus from a perisinous abscess finds its way through the posterior condyloid canal, there is edema and tenderness under the occiput, in the upper third of the posterior cervical triangle.

Gerhardt observed that when there is a thrombus in the sigmoid sinus and bulb, the external jugular vein empties more easily into the collapsed

internal jugular, than into the normally filled internal jugular on the unaffected side. Consequently the external jugular vein on the normal side appears fuller than that on the affected side. This is not a very reliable diagnostic sign, however, for two reasons. The external jugular empties into the innominate vein more frequently than into the internal jugular. When the thrombus extends into the internal jugular vein to a point below where the external jugular joins the latter, the external jugular on the affected side will be fuller than on the normal side.

If a stethoscope is placed over the internal jugular vein, under normal conditions, a bruit can be heard, due to the passage of blood through the vein. If pressure is made over one internal jugular vein without compressing the carotid artery, there is an increased rapidity of the blood flow through the other internal jugular, and the bruit over the latter becomes more distinct. When there is an obturating thrombus in the sigmoid sinus or bulb, there is little or no blood passing through the internal jugular vein of the affected side, and the normal bruit cannot be heard on that side.

Crowe observed that when both internal jugular veins are obliterated by compression, there is marked stasis of the intracranial venous circulation. This manifests itself in the eye as a dilation of the retinal veins, which can be readily seen with the ophthalmoscope. When there is congestion in the intracranial venous system, the anastomotic vessels between the intracranial and extracranial venous systems become engorged. One of these anastomotic veins, which can be seen on the surface, is the supraorbital vein. This vein forms a communication between the cavernous sinus and the facial vein. When both internal jugular veins are compressed, the supraorbital veins become dilated. If pressure is released on one internal jugular vein, the dilated veins in the retina and in the face will immediately return to normal size, on account of the free communication between the two internal jugulars.

This explains why eyeground changes are not present in every case of obturating sinus thrombosis.

When there is an obturating thrombus in one sigmoid sinus or jugular bulb, pressure on the internal jugular vein of the opposite side produces engorgement of the retinal and supraorbital veins of both sides. Pressure on the internal jugular vein of the affected side produces no changes in the retinal and supraorbital veins.

The test is no longer positive, four to six weeks after ligation of the internal jugular vein, on account of the establishment of collateral circulation.

When thrombophlebitis of the sigmoid sinus extends down to the internal jugular vein, there is usually an inflammation of the tissues around the jugular vein. This is especially true of the lymph glands which surround the vein. Inflammation of these lymph glands and of the perijugular connective tissue may give rise to a tender swelling along the jugular vein. There may be pain in this region on turning the head. The hard cord-like feeling of a thrombosed internal jugular vein, which has been described by some otologists, is rarely present.

When there is swelling of the lymph glands at the side of and behind the posterior pharyngeal wall, there may be pain on swallowing.

A cervical adenitis due to other causes cannot be differentiated from the adenitis which is caused by phlebitis of the internal jugular vein. Sometimes the phlebitis of the vein is secondary to the adenitis.

Extension of the thrombus from the internal jugular vein to the facial vein occasionally gives rise to edema of the face or abscess of the face.

Extension of the thrombus from the internal jugular to the thyroid vein may cause a perichondritis of the larynx or an abscess of the thyroid gland.

Test for Sinus Thrombosis. A test for the determination of sinus thrombosis based upon changes in the spinal fluid pressure was described by

Dowman, Tobey and Ayer, and Bruenings and Perwitzschky. They all arrived at the same conclusions independently of one another.

The test is based upon the Queckenstedt phenomenon. Queckenstedt found that by compressing both internal jugular veins in the neck, the spinal fluid pressure was increased. This increase in the spinal fluid pressure is due to an increase in the intracranial pressure, caused by the venous engorgement within the skull. This phenomenon was utilized to diagnose a block in the spinal canal from tumor or inflammatory adhesions. If pressure on both internal jugulars caused no rise in the spinal fluid pressure, a diagnosis of a block in the spinal canal could be made.

This test is now utilized to determine the presence of a thrombus in the lateral sinus, jugular bulb or internal jugular vein.

If there is a thrombus in the sinus, bulb or vein, pressure on the internal jugular vein on that side will produce no increase in the spinal fluid pressure, because the pressure on the vein does not cause intracranial venous engorgement. Each vein is compressed separately, and the effects on the spinal fluid pressure compared.

The test is of value, not only to diagnose the presence of a sinus thrombosis, but also to indicate which side is involved.

The test is described by Tobey and Ayer:

"Lumbar puncture is done with the patient in the lateral position, and the spinal fluid is allowed to run into a manometer of 2 mm. bore. The initial pressure is noted, also the presence of pulse and respiratory oscillations, as evidence of the patency of the manometric system. An assistant presses on one side of the neck between the larynx and the sternocleidomastoid muscle, until he feels a strong carotid arterial pulsation. This procedure is repeated on the opposite side of the neck. Then for comparison, both sides of the neck are pressed simultaneously. In a typical case of sinus thrombosis, when the jugular on the normal side is pressed, there is a rapid

rise in pressure to two or three times the initial reading. This rise is equal to that when both jugulars are compressed. Pressure of the jugular on the diseased side causes no rise, or a slow rise of 10 to 20 mm."

The possible fallacies of the test, according to Tobey and Ayer are:

"1. Absence of rise or very slight rise occasionally occurs without obstruction in either venous or subarachnoid fluid system, when the cerebrospinal fluid is under low tension. If 10 to 20 c.c. of salt solution are introduced into the spinal canal, the dynamic tests will appear normal.

"2. In persons with thick necks, it may be very difficult to locate and compress the veins.

"3. Abnormal rise in pressure may result from carelessness in performing the test. If the patient coughs, holds his breath, grunts or even moves his head or body, there will be a rise of pressure.

"4. There may be normal differences on the two sides. A difference of over 50 mm. is unusual, and over 100 mm., exceptional. With a normal initial pressure of 150 mm., the rise on jugular compression is seldom less than 50 mm. usually over 100 mm. The rise in fluid pressure seems to be somewhat less, when the vein on the dependent side is compressed. It is likely that this is due to difficulty in reaching the vein, rather than an inherent difference in physiology.

"5. Anatomic variations in the size of the lateral sinuses are possible, but rare."

The dangers of the test, according to Tobey and Ayer are:

"1. Sudden death from the presence of subtentorial abscess or tumor, following lumbar puncture.

"2. Precipitation of acute meningitis."

"The first danger may be eliminated by careful preliminary examination of the patient. The second danger relates to liberation of organisms from an area of localized infection, or from the blood stream. The former appears

unlikely, unless large amounts of fluid are withdrawn. The latter rests on experimental evidence by Weed, in which it was shown that in the presence of a blood infection, the passage of microorganisms from vessels to meninges may be facilitated by withdrawal of cerebrospinal fluid, causing cerebral venous congestion. But this danger is very slight."

No ill effects were seen in 22 cases examined by Tobey and Ayer by this method.

Perwitzschky modified the test to determine the presence of a partial stenosis in the sinus, such as is caused by a mural thrombus.

The normal spinal fluid pressure is 180 to 260 mm. On compression of the jugular it rises to from 360 to 420 mm. A change in the maximum height of the spinal fluid pressure does not occur when there are variations in the lumen of the sinus or vein. But if a vessel is compressed, and the pressure is released, the initial blood pressure in a vessel of larger caliber is re-established more quickly than in one of smaller caliber. In other words, a larger vessel refills more quickly than a smaller vessel. Consequently the re-establishment of spinal fluid pressure, after pressure on the jugular vein is released, occurs more quickly when the sinus has a normal lumen than in cases where the sinus is contracted by a mural thrombus.

The technique of the test, as described by Perwitzschky is as follows: "The patient lies in a horizontal position on his side, with his head a continuation of the line of the spinal column. Lumbar puncture is done. When the needle lies in the spinal canal, and the height of the fluid in the manometer is noted, both jugulars are compressed at the same time. The increase in the height of the fluid is noted, in order to exclude a block in the cisterna or the spinal canal. One vein is then compressed from behind the patient, in the carotid triangle. Four fingers are used, the thumb resting on the cervical vertebrae for support. Pressure must be only from before backward. An assistant with a stop-watch calls off every five seconds, and the manometer

reading is made at intervals of five seconds. When the maximum height is reached, compression is maintained for twenty seconds, and then released. The manometer is again read every five seconds. After twenty seconds a similar test is made on the other vein."

When both sinuses are normal, the difference between the two sides, in the time of fall to the normal, after releasing pressure on the vein may be ten seconds. In a case of perisinous abscess, with compression of the sinus, examined by Perwitzschky, the return to normal spinal fluid pressure was thirty seconds on the normal side and seventy-five seconds on the diseased side, a difference of forty-five seconds. In a case of mural thrombus in the sigmoid sinus, the return to normal pressure was twenty seconds on the normal side and seventy seconds on the diseased side, a difference of fifty seconds.

Brünings found that the pressure of the spinal fluid is not the same as the intracranial pressure. His method of measuring the intracranial pressure is based upon the following facts: The brain may be compared to a blood-filled sponge, in which the brain tissue is incompressible, but which can be emptied of part of its blood, or in which the blood-content can be increased. The arterial blood enters the skull with rhythmic pulsations. It leaves the skull through the veins, in a continuous stream or with very slight pulsations. The entire mass of cerebral blood, and therefore the brain volume, undergoes pulsation, which can be seen in an exposed portion of the dura. These dural pulsations are due to a volume impulse of the brain and have nothing to do with blood-pressure. Whether the dural pulsations are large or small depends upon the tension of the dura. The more tense the dura the less marked are the transmitted pulsations from the brain. The extent of the dural oscillations, therefore, is a measure of the dural tension. If the brain pressure from within the dura is gradually neutralized by an increasing manometer pressure from without, the time when both pressures become equal will be marked by the time when the dural oscillations will have

reached their maximum, as the dura will then have no tension whatever. In other words, when the pressure produced on the outer surface of the dura by the manometer equals the pressure produced on the inner surface of the dura by the brain, there will be no tension whatever on the dura.

A capsule is placed over the dura in a trephine opening. It is connected by a tube to a manometer. Between the manometer and the capsule a pressure bulb is interpolated. The manometer registers the dural oscillations and the pressure in the capsule. As the pressure in the capsule is increased by the bulb the dural oscillations increase in size, at first, and later decrease. When the oscillations reach their maximum, the pressure in the manometer indicates the brain pressure. The respiratory movements of the dura are sometimes confusing in reading the pulsatory oscillations.

In this manner, Brünings found that the average intracranial pressure was 240 mm. of water. The intracranial pressure is independent of the general blood pressure. He found that when there is unilateral compression of the jugular vein, the cerebral pressure increases, and that it increases still more when there is pressure on both jugulars. The cerebral pressure is never as high as the arterial blood pressure, because even with pressure on both jugulars some venous channels remain open, for example, the veins in the spinal canal. The highest recorded cerebral pressure was 750 mm. of water. He found that the normal oscillations of brain pressure which are synchronous with respiration, stop when the jugular veins are compressed. They are therefore dependent upon the exspiratory stasis of the venous outflow from the skull. He found that the pressure of the spinal fluid is rarely the same as the intracranial pressure. Either may be higher or lower than the other. The difference may be more than double. The increase of intracranial pressure due to jugular compression, however, is transmitted to the spinal pressure. The increase in the spinal pressure reaches its maximum about sixty seconds after the increase in the intracranial pressure.

When there is marked lowering of cerebral pressure from excessive loss of spinal fluid following lumbar puncture, there may be symptoms of collapse. These symptoms can be prevented by jugular compression.

Examination of the spinal pressure is so much easier than examination of the cerebral pressure, that, even though the former is a roundabout method, and the results are less accurate and more subject to error than in the latter method, it will probably always be the method of choice.

Symptoms Due to Interference with Cerebral Circulation and Extension of Inflammatory Process. The local symptoms of sigmoid sinus thrombosis which are due to interference with the cerebral circulation and to extension of the inflammatory process to the adjacent brain, membranes and nerves, are headache, signs and symptoms of inflammation of the brain and meninges, and involvement of the optic nerve and the sixth nerve.

The headache which occurs in sinus thrombosis is not characteristic. It may be dull or sharp, constant or intermittent, and may be located in any part of the head. It is usually due to circulatory disturbances in the brain. The circulatory disturbances result in venous engorgement of parts of the brain. This, in turn, may cause interference with the normal outflow of the cerebrospinal fluid from the skull, so that symptoms of serous meningitis may supervene.

When the infective process extends from the sigmoid sinus to the meninges and brain tissue, symptoms of meningitis or brain abscess occur.

Optic Nerve Changes. Optic nerve changes are fairly common in sigmoid sinus thrombosis.

White examined the eyegrounds in 62 cases of sinus thrombosis. Normal eyegrounds were found in 29 of these cases. The remaining 33 cases showed some changes in the optic nerve. He groups the cases according to the severity of the eyeground changes into four classes:

1. Dilatation of the retinal veins.
2. Blurring of the disc margins.
3. Slight papilledema (under two diopters).
4. Marked papilledema (over two diopters), usually with hemorrhages.

Of the 33 cases with eyeground changes, 4 had dilatation of the retinal veins; 13 had blurring of the disc margins; 8 had slight papilledema; and 8 had marked papilledema. In none of these cases was the vision markedly impaired.

Atkins observed 40 cases of lateral sinus thrombosis. Eyeground changes were noted in 4 cases before operation, and in 13 cases after operation.

Eves reported 38 cases. There were eyeground changes in 10 per cent.

Ruttin observed 30 cases of sinus thrombosis. There were 8 cases of papilledema. Of these, 6 occurred after jugular ligation and operation on the sinus. The papilledema came on from two days to one month after the operation.

Blau observed 162 cases of uncomplicated sinus thrombosis. He found vascular changes in the retina in 20 (12 per cent); optic neuritis in 17 (11 per cent); and choked disc in 13 (8 per cent), making a total of 50 (31 per cent).

The optic-nerve changes in sinus thrombosis are due to mechanical causes, and are not inflammatory. In inflammation of the optic nerve there is usually marked impairment of vision, whereas in the papilledema which occurs in sinus thrombosis the vision is normal or only very slightly reduced. The fact that optic-nerve changes come on more frequently after operation on the sinus and jugular ligation than before operation militates against the assumption that they are inflammatory, as the inflammatory changes about the sinus usually improve after operation.

Ruttin believes that the papilledema is due to stasis in the petrosals and the cavernous sinus, with resultant stasis in the central retinal vein.

This is unlikely, because in cavernous sinus thrombosis, when the cavernous sinus is completely blocked, optic-nerve changes are not more common than in thrombosis of the lateral sinus.

The changes in the optic nerve are in all probability due to circulatory changes in the brain and membranes causing an increase in the cerebrospinal fluid. This increase of fluid extends into the subarachnoid space about the optic nerve, and interferes with the normal lymph flow in the perivascular lymph spaces in the nerve, so that edema of the nerve results. Some authors believe that in addition to the mechanical factor, there is a toxic element in these cases. By injecting salt solution into the subdural space, and thus increasing the subdural pressure, edema of the optic nerve was produced experimentally. Clinical evidence of the influence of pressure in these cases is furnished by the rapid subsidence of the papilledema, in most cases, following decompression.

When papilledema is present, it is not safe to do a lumbar puncture, as death may result from herniation of the brain into the foramen magnum. It is best to do a preliminary puncture of the lateral ventricle through a small trephine opening in the parietal eminence, in order to reduce the intracranial pressure. Then lumbar puncture can be performed in safety. Inflammation of the sixth nerve sometimes occurs with sinus thrombosis. It may occur before or after operation on the sinus and jugular. It is probably not due to circulatory changes in the brain, but to a localized meningitis at the base, or to a necrosis of the apex of the petrous pyramid. The paralysis of the nerve usually clears up in a few weeks.

Peribulbar Abscess. The local symptoms of thrombosis of the jugular bulb are due to peribulbar inflammation or peribulbar abscess. There can be no extension from the bulb to the brain or meninges, unless the peribulbar abscess extends upward and becomes a perisinous abscess.

When pus from a peribulbar abscess finds its way through the posterior condyloid canal, it may produce a painful swelling in the upper part of the posterior cervical triangle.

When the peribulbar abscess extends downward along the sheath of the internal jugular vein, it causes a perijugulitis. This manifests itself by the presence of swelling and tenderness along the course of the jugular vein in the neck, pain on turning the head, and sometimes pain on swallowing.

Peribulbar abscess may give rise to an inflammation of the ninth, tenth and eleventh nerves, as they pass through the jugular foramen.

Involvement of the glossopharyngeal nerve may cause paralysis of the muscles of deglutition, pain on swallowing, or paralysis of the soft palate. Involvement of the vagus may cause slow pulse, hoarseness, aphonia and dyspnoea. It may result in death from respiratory paralysis. Involvement of the spinal accessory nerve may give rise to spasms of the sternomastoid and trapezius muscles.

When there is a phlebitis of the anterior condyloid vein, it may cause an inflammation of the hypoglossal nerve, with paralysis of the tongue.

In many cases it is impossible to tell whether the thrombus is located in the sigmoid sinus or in the bulb, before operation.

The findings at operation sometimes help in the diagnosis. In many cases, the appearance of the sinus wall gives no information as to the contents of the sinus.

Appearance of Sinus. A normal sinus wall is bluish-gray in color. The surface is slightly glistening. It is easily compressed. The feeling given to the finger is like that of a bladder moderately distended with water.

When a perisinous abscess is present, the surface of the sinus is covered by granulations. It is impossible to tell in these cases, either by inspection or by feeling, whether a thrombus is present or not. Granulations on the outer surface of the sinus occur more frequently without than with thrombosis.

The granulations offer a protection against invasion of the interior of the sinus by the infection.

Sometimes there is a fistula in the outer sinus wall and pus can be seen to exude from the interior of the sinus. In such cases, the diagnosis is evident.

The sinus wall may be normal in color and appearance, and yet the sinus may be the seat of an obturating thrombus.

In some cases of sinus thrombosis, the outer wall of the sinus is white and opaque. Sometimes the wall has a greenish or brownish discoloration. When there is a thrombus in the sinus, it may have a doughy feel, or it may feel firm and resistant.

The sinus wall rarely shows pulsations or respiratory movements. When a considerable portion of the sinus wall and adjacent dura is uncovered, it may show pulsations which are transmitted from the brain. They are not a venous pulse. The presence or absence of a thrombus in the sinus has no effect upon these transmitted brain pulsations. The brain pulsations are dependent upon the pulsation of the large arteries which the brain contains.

There is normally a venous pulse in the superior vena cava, which is sometimes transmitted to the lower end of the internal jugular vein. The cause of the venous pulse is not very clear, but it is probably due to contraction of the muscular fibers in the wall of the vena cava. The pulse occurs just before the auricular systole and probably helps in filling the auricle with blood. This venous pulse is not transmitted to the lateral sinus, as is evidenced by the fact that when the sinus is incised, the blood-flow from the wound is steady and not intermittent. Mann, however, observed that a real venous pulse could be produced in the sinus, when the patient is placed in the upright position and the head is turned about a vertical axis so that the mastoid process of the side to be examined is brought to lie above the sterno-clavicular articulation of the same side. In this position, the neck muscles exert no pressure on the jugular vein and the walls of the vein are

held widely separated. When such a venous pulse occurs, it indicates that there is no thrombotic obstruction between the sinus and the heart.

Respiratory movements of the sinus are uncommon. When they do occur, there is a collapse of the sinus during inspiration and a filling up of the sinus during ex-spiration. The negative pressure in the chest during inspiration sucks the blood out of the sinus, and the positive pressure in the chest during ex-spiration offers a resistance to the flow of blood from the sinus to the chest, so that the sinus is allowed to fill up. Respiratory movements in the sinus are favored by a sudden deep inspiration, by the sitting position and by swallowing. They are especially apt to occur when the sinus is plugged peripherally and is free centrally.

If respiratory movements are present during operative procedures on the sinus, there is danger of air embolism. This may be prevented by plugging off the lower end of the sinus, or by pressure on the internal jugular vein in the neck.

Sometimes, when the sinus plate is removed, the outer sinus wall is found to be collapsed. This indicates that there is an occluding thrombus in the sinus peripheral to the collapsed area.

In some cases, the destruction of the outer sinus wall may be so extensive as to mislead the operator into thinking that the visceral is the outer wall of the sinus.

If the emissary is thrombosed, it usually indicates that the sigmoid sinus is thrombosed, but not necessarily so.

Aspiration of the sinus by means of a needle does not always give reliable information as to the contents of the sinus. Fluid blood may be drawn into the syringe, and yet the sinus may be the seat of a mural thrombus; or there may be an obturating thrombus lower down in the sinus, or higher up. If nothing is drawn into the syringe, we assume that there is an obturating thrombus in the sinus. We are not always sure, however, that

the point of the needle is in the lumen of the sinus. The point of the needle may be in the visceral wall of the sinus. This is especially apt to occur when the lumen of the sinus is narrowed by the pressure of a perisinous abscess. If the outer wall of the sinus is very much thickened by granulation tissue, the point of the needle may not enter the lumen of the sinus. We may think it is in the lumen, but it may be only in the outer wall of the sinus. If the needle enters the visceral wall of the sinus it may carry infection into the meninges or the brain. In order to avoid injuring the visceral wall of the sinus the needle should be introduced very obliquely. If pus or a discolored fluid is drawn into the syringe the diagnosis of a broken-down thrombus is positive.

More reliable information can be obtained by incision of the outer sinus wall. The entire width of the sinus should be uncovered for a distance of about 1 inch in the region of the knee. The lumen of the sinus should be obliterated above and below, by means of an iodoform gauze plug which is inserted between the outer sinus wall and the overlying bone. If these plugs are properly applied to cover the entire width of the sinus, there will be complete control of bleeding when the sinus is opened. Care must be taken to block off the entrance of the superior petrosal sinus. A longitudinal incision is now made into the outer sinus wall between the two plugs, large enough so that the interior of the sinus can be carefully examined. If an obturating thrombus is present in this location it can be readily seen. Even a mural thrombus is easily discovered, as the view is not obscured by bleeding. If no thrombus is found at the site of incision, the upper plug is removed. If there is free bleeding, it is an indication that there is no thrombus above the knee of the sinus. If only a small stream of blood follows removal of the upper plug, it is an indication that there is a mural thrombus in the upper part of the sinus. The upper plug is then replaced and the lower plug removed. If there is free bleeding from the lower end it shows that the

bulb is probably free. It is possible, however, for a thrombus to be present in the lower part of the bulb, as the bleeding may come from the inferior petrosal sinus, or from the condyloid vein, or both.

If no bleeding follows removal of the lower plug it is proof that there is an obturating thrombus in the bulb. If pus wells up from the lower end of the sinus, it shows that there is a broken-down thrombus in the bulb.

Tobey has demonstrated a thrombus in the sigmoid sinus "by incising the emissary vein, having first passed a ligature around the vein, proximal to the point of incision, and then inserting a small silver probe into the sinus. The use of the probe is necessary, since the valve-like opening of the emissary into the sinus often prevents the flow of blood from the sinus into the vein (the normal direction of the flow being from the emissary into the sinus)."

If it is shown that there is no thrombus in the sinus or bulb, the two compression plugs are removed and the bleeding from the sinus is controlled by iodoform gauze packing over the sinus wound. It is usually not necessary completely to obliterate the lumen of the sinus by the packing in order to control bleeding. If the intervention is done under strictly aseptic precautions, the wound will usually heal without thrombus formation, and without obliteration of the sinus. If there are bacteria in the circulation, however, a thrombus is very apt to form at the site of incision. This would have to be removed at a secondary operation.

A diagnosis of bulb thrombosis can rarely be made without exploratory operation. If, when the sigmoid sinus is incised, there is no bleeding from below, or if pus wells up from below, we can make a positive diagnosis of bulb thrombosis. If only a small stream of blood comes up from below, there may be a mural thrombus in the bulb, or there may be an obturating thrombus in the lower part of the bulb, the bleeding coming from the inferior petrosal sinus or the posterior condyloid vein.

If the internal jugular vein is ligated above the entrance of the facial vein into the jugular, and the latter is empty above the ligated point, we can make a diagnosis of bulb thrombosis.

A bulb thrombosis may be diagnosed by Whiting's test. It is described by Whiting as follows: "The left index finger is placed across the lower end of the sinus with sufficient pressure to obliterate it. The right index finger is placed above the left, and with a milking motion passed up to the knee of the sinus, emptying the sinus between the two fingers. An assistant applies pressure to the jugular vein in the neck to intensify the strength of the blood-stream from below. If the lower finger is removed, and the sinus refills, there is no obstruction in the bulb or jugular vein. If the sinus does not refill, there is a thrombus in the bulb."

If there is a thrombus in the sigmoid sinus, a manipulation such as is described above might result in the breaking off of a portion of the thrombus, and consequently allowing it to enter the general circulation.

Determination of Side of Thrombus in Double Mastoiditis. When both mastoids are involved and there is evidence of a sinus thrombosis, it is often difficult to determine on which side the thrombus is located. The following points in the diagnosis, as tabulated by Chase, are of value in this regard:

1. Tobey's test. A manometer is attached to the spinal puncture needle, and the spinal pressure is noted before and after compression of either internal jugular vein. Compression of the jugular on the normal side will cause an increase in the spinal fluid pressure. Compression of the jugular on the side of the thrombus has no effect on the spinal pressure.

2. Crowe's test. Compression of the internal jugular vein on the side opposite the thrombus causes an increase in the fullness of the retinal and supraorbital veins.

3. The roentgen-ray picture may show greater disintegration of the mastoid on one side. The picture may show a perisinous abscess. The side

which has the greatest amount of destruction in the mastoid is likely to be the side on which the sinus is involved.

4. The mastoid which was involved first is more likely to be the side of the thrombosis.

5. A comparison of the tenderness and glandular involvement along the jugular vein on the two sides may give some information.

6. The extent of the bone destruction shown at the mastoid operation may be a guide, especially if the bone destruction extends to the wall of the sinus in one mastoid and not in the other.

7. The appearance of the sinus wall sometimes helps. Granulations on the sinus wall have no significance. Discoloration of the wall is more significant. A fistula in the sinus wall is the only positive diagnostic sign.

8. Aspiration of both sinuses may help.

9. Comparison of the bacterial content of the two sinuses may be of value. The side which contains the most bacteria may be the side which contains the thrombus.

10. Whiting's test. This is of diagnostic value only if the thrombus is in the lower end of the sigmoid sinus or bulb, and if it is obturating.

Barnhill states that when there is a double mastoiditis, with sinus thrombosis on one side, the bone bleeds more at operation on the side which has the thrombosis. This is due to the venous stasis in the bone, which results from the thrombus.

The external local signs which may be present when the thrombus is located in the superior longitudinal sinus are dilatation of the veins of the scalp, and a swollen tender area in the region of the parietal emissary.

The symptoms due to circulatory disturbances in the brain with thrombosis of the superior longitudinal sinus or straight sinus are usually very prominent. There is usually severe headache. There may be delirium

and localized or general convulsions. There may be stupor. Aphasia, hemiplegia or crossed facial paralysis may occur.

Doyle drew the following conclusions from a review of the literature of case histories of thrombosis of the superior longitudinal sinus:

"1. In the absence of meningitis, the early onset of apathy or stupor in a patient with evidence of sinus thrombosis generally indicates retrograde extension to the superior longitudinal sinus, especially if associated with choked discs and convulsions.

"2. When tumor or inflammatory disease of the brain can be excluded, Jacksonian seizures showing progression from one foot to the other, or beginning in the foot and gradually involving the homolateral upper extremity, suggest impairment of the circulation of the cerebral veins, and probable thrombosis of the superior longitudinal sinus.

"3. Abrupt onset of symptoms of increased intracranial pressure which follow a fluctuating course should bring to mind the possibility of thrombosis of the superior longitudinal sinus or ventricular tumor. The absence of the element of progression after a fair length of time, or actual regression of symptoms is suggestive of thrombosis of the superior longitudinal sinus. If the syndrome is associated with edema of the eyelids and dilatation of the veins of the eyelids and forehead, and if fracture of the skull, orbital and peri-orbital infections and thrombosis of the cavernous sinuses can be excluded, the diagnosis is established."

Hessler in 1895 collected from the literature 6 cases of infective thrombosis of the superior longitudial sinus. In addition, 2 cases were reported by Carli, 2 by Gradenigo, 1 by Richter, 1 by Hoelscher, 1 by Eagleton, 1 by Lermoyez, 1 by Ruttin, 1 by Hirsch and Schwab and 3 by Lillie.

The occurrence of cerebral symptoms in uncomplicated sinus thrombosis may be due to:

1. The toxic effect of bacteria in the blood.

2. Disturbances of the blood and lymph circulation within the skull.
3. The thrombus in the sinus.
4. Retrograde thrombosis in the cerebral veins.

Local Symptoms in Cavernous Sinus Disease. The local symptoms due to circulatory disturbances in cases of cavernous sinus thrombosis are edema of the lids and conjunctiva, exophthalmos, choked disc, swelling of the forehead, nose and cheeks, and epistaxis. In order to produce sufficient orbital venous engorgement to cause exophthalmos, the thrombus must extend into the ophthalmic vein, as the orbital veins anastomose freely with the facial vein. According to Eagleton, post-mortem examination shows that exophthalmos is not caused by venous engorgement alone, but also by inflammatory infiltration of the orbital tissues which surround the thrombosed veins. Orbital abscesses, due to perforation of one of the thrombosed veins in the orbit are not uncommon. These inflammatory changes in the orbit are especially likely to occur in those cases of cavernous sinus thrombosis which are due to extension from the face, because the orbital veins are always thrombosed in these cases.

Chemosis or edema of the conjunctiva is frequently so marked, that some of the conjunctival tissue extends through the palpebral fissure even when the lids are closed. The swollen conjunctiva may partly cover the cornea.

Swelling of the lids, especially of the upper lid, usually causes complete closure of the eye. It often makes it difficult to determine whether ptosis is present. Edema of the forehead, nose and cheeks, as a rule is not as marked as in the lids. The edema of the conjunctiva, lids and face is due to the same causes which produce the exophthalmos, namely, venous engorgement and cellulitis.

The thrombus in the cavernous sinus and the inflammation in the wall of the sinus usually give rise to an inflammatory process in the nerves

which lie in the wall of the sinus, that is, the third, fourth, the first division of the fifth, and the sixth nerves. The symptoms which are caused by inflammation of these nerves are paralysis of the ocular muscles, with strabismus, ptosis and pupillary changes. When all the muscles are involved, there may be complete immobility of the eyeball. The pupil is usually dilated and immobile. Inflammation of the first division of the fifth nerve causes neuralgic pain in the area of distribution of this nerve in the forehead, about the eye and in the nose. In some cases, there is an extension of the inflammatory process from the cavernous sinus to the Gasserian ganglion. When this occurs, there may be symptoms typical of trigeminal neuralgia. Herpes of the face occurs frequently as the result of irritation of the Gasserian ganglion.

The symptoms of cavernous sinus thrombosis usually begin on one side of the head, and in a few days they appear on the other side of the head. This is due to the fact that a thrombus in one cavernous sinus rapidly spreads through the circular sinus to the opposite cavernous sinus. This is one means of differentiating between the exophthalmos of cavernous sinus disease and that due to orbital cellulitis or to frontal and ethmoidal sinusitis. In the latter conditions, the disease almost always remains unilateral.

When there is extension of the thrombus from the cavernous sinus to the pterygoid plexus, there may be edema of the pharynx; edema of the face may result from extension of the thrombus into the facial vein. Extension of the inflammation from the cavernous sinus to the meninges or brain may result in meningitis or brain abscess.

When there is retrograde extension of the thrombus from the cavernous sinus to the cerebral veins, there may be focal brain symptoms corresponding to the portion of the brain which is involved.

Eagleton divides the cases of cavernous sinus thrombosis into two classes, the acute and the chronic forms. The acute cases give the classical picture with symptoms due to sudden blocking of the veins which empty into the

cavernous sinus. In the chronic cases there is a slow gradual occlusion of the sinus. There are no symptoms due to venous engorgement, because there is time for collateral circulation to become established. In the chronic cases there are few or no localizing symptoms. Hölscher collected 11 cases of cavernous sinus thrombosis without exophthalmos, ocular paralyses or eyeground changes. Cavernous sinus thrombosis due to suppuration of the petrous pyramid is usually of the slow compensatory type. In these cases there are symptoms of chronic sepsis, irregular chills, malaise, headache and night-sweats. Occasionally there is a sixth nerve paralysis. Exophthalmos is rare, except as a very late development.

Changes in the eyegrounds occur in cavernous sinus thrombosis about as frequently as in thrombosis of the lateral sinus.

A thrombus may extend from the cavernous sinus into the ophthalmic veins without causing eyeground changes, on account of the free anastomoses between the ophthalmic veins and the facial veins and pterygoid plexus. When the central retinal vein becomes involved, or when the collateral circulation becomes seriously hampered, eyeground changes may occur.

Abscesses due to retrograde thrombosis in the venous radicles of the cavernous sinus may occur in the brain, the orbit, the scalp, the face or the nasopharynx.

Early Symptoms. The early symptoms of cavernous sinus thrombosis, in the order of their appearance, are, according to Langworthy:

1. Deep seated pain about the eyes.
2. Septic temperature.
3. Slight orbital congestion.
4. Lachrymation.
5. Conjunctival edema.

6. Lid-swelling.
7. Ptosis.
8. Exophthalmos.

Pupillary changes, papillary edema, meningeal or brain symptoms and metastases appear later.

When there is extension of the thrombotic process from the lateral sinus to the petrosal sinuses, there may be no additional symptoms. Sometimes, however, this extension is marked by a dilatation of the veins in the temporal region and in the retina, the occurrence of epistaxis, and the onset of epileptiform seizures.

Usually there are no signs of the extension until the thrombus reaches the cavernous sinus. In some cases of thrombosis of the superior petrosal sinus, there is trigeminal neuralgia.

In marantic sinus thrombosis, there are no septic symptoms; no fever, no chills or sweats, no rapid pulse and no bacteriemia. There is dilatation of the frontal and parietal veins, with edema of the soft tissues of the frontal and parietal region of the scalp. There may be epistaxis, strabismus, contractions and rigidity of the extremities, neck and back; localized or general convulsions; delirium, stupor or coma. Occasionally there is paralysis of one or more of the extremities, or aphasia. The symptoms are those due to intra- and extra-cranial venous congestion, with resultant disturbances of brain function and may be temporary. They may disappear when collateral circulation is established.

Types of Otogenic General Sepsis

Cases of otogenic general sepsis can be divided into two types, the septic and the pyemic types. To these may be added a third type, which is not really a form of otogenic general sepsis, but a sinus thrombophlebitis without general sepsis, that is, the form of thrombophlebitis which runs its course

without symptoms, and is discovered accidentally in the course of an operation on the mastoid.

The *septic* form of otogenic general sepsis runs a very stormy course, with severe general symptoms, very high continuous fever, and rapid death. There is marked anemia, restlessness, disturbances of the sensorium, headache, loss of appetite, dry tongue, tympanites, constipation or diarrhea, rapid pulse, enlargement of the spleen and albuminuria. There are no metastases. In the milder cases of the septic form of this disease, there may be recovery by lysis.

The *pyemic* form of otogenic general sepsis is characterized by remittent or intermittent temperature, chills followed by sweating, and metastases.

Most of the cases are of a mixed septico-pyemic form, with either the pyemic or the septic element in the foreground.

The septic type of the disease frequently occurs with phlebitis without thrombus formation. This occurs when there is a pathologic filterability of the vessel walls for microorganisms. In this type of the disease, there may be septic endocarditis, hemorrhages into the muscles, retina and chorioid, septic nephritis, hepatitis with icterus and enteritis. Paralysis of the functions of the heart and brain predominate in the picture, and cause death.

Diagnosis

The diagnosis of sinus thrombophlebitis is based upon four groups of symptoms: (1) The pyemic blood poisoning; (2) Signs of obstruction of the blood vessels; (3) Signs due to extension of the inflammation to neighboring structures; (4) Metastases.

1. The pyemic signs are the characteristic fever with chills, large spleen and changes in the blood.

2. The signs of obstruction of the blood vessels are Griesinger's and Gerhardt's signs, Crowe's and Tobey's signs, and exophthalmos.

3. Signs due to extension of the inflammation to neighboring structures are the evidences of involvement of the ninth, tenth, eleventh and twelfth cranial nerves.

Differential Diagnosis

Some of the diseases from which sinus thrombophlebitis must be differentiated are malaria, typhoid fever, erysipelas, acute miliary tuberculosis, septic endocarditis, pneumonia, orbital cellulitis, acute tonsillitis, cervical adenitis, nasopharyngeal infections and pyelitis.

It is necessary to differentiate between *malaria* and sinus thrombosis, when the malaria is complicated by a middle-ear suppuration. The type of temperature, with chills and sweats, is the same in both diseases. An examination of the blood in malaria will show the plasmodium. In sinus thrombosis, blood culture may show a bacteriemia. There is some enlargement of the spleen in both conditions. The period of normal temperature is likely to be longer in malaria than in sinus thrombosis, and the patient is likely to feel more normal in malaria during the time when the temperature is down.

Typhoid fever complicated by middle-ear suppuration is occasionally confused with sinus thrombosis. The temperature in typhoid fever is sometimes remittent. The temperature in sinus thrombosis may be continuously high. There may be enlargement of the spleen and headache in both conditions. The roseolar eruption on the abdomen in typhoid may help to differentiate it from sinus thrombosis. A blood culture will usually decide the diagnosis. In typhoid fever the blood contains typhoid bacilli, whereas in sinus thrombosis the blood usually contains streptococci. Typhoid bacilli as a rule can be demonstrated in the blood fairly early in the disease.

Erysipelas is sometimes mistaken for sinus thrombosis. Erysipelas not infrequently develops in the skin about the ear, a few days after a mastoid operation. The temperature in erysipelas is usually very high, and may be continuous, remittent or intermittent. If the redness in the skin appears

within a day or two after the onset of the high temperature, the diagnosis is easy. But sometimes the redness does not appear for five, six or seven days after the onset of the fever. In these cases the diagnosis is very difficult. A blood culture may be of assistance but, although a bacteriemia is uncommon in erysipelas, it does sometimes occur. Tobey's test may help, if there is an obturating thrombus.

The writer recalls a case with a remittent temperature going up to 105° F. every day for five days. He finally decided to explore for a possible sinus thrombosis, when the rash of an erysipelas appeared. Heine and Leutert each saw a case of erysipelas, in which the rash did not appear until the temperature had lasted eight days.

Acute miliary tuberculosis may be confused with sinus thrombosis, especially when the latter condition is complicated by multiple pulmonary metastases. A blood culture helps in these cases. Examination of the eye-grounds may be of assistance, as solitary tubercles may be seen in the chorioid.

Septic endocarditis may be confused with sinus thrombosis. Septic endocarditis is sometimes a complication of sinus thrombosis. There is a bacteriemia in both conditions. When septic endocarditis results from sinus thrombosis, the former may cause a persistence of the bacteriemia after the thrombus has been removed from the sinus. The physical signs over the heart and the character of the pulse may aid in the diagnosis.

Pneumonia may have to be differentiated from sinus thrombosis. When there are pulmonary metastases in sinus thrombosis, the physical signs in the chest are confusing. Pneumonia may be present with very few or no physical signs in the chest. The bacteria in the blood in pneumonia are pneumococci, whereas in sinus thrombosis, pneumococci scarcely ever occur in the blood. A roentgen-ray picture of the chest often gives valuable information. The writer saw 2 cases of pneumonia in children, 1 case coming on

after mastoid operation, and the other occurring with an acute middle-ear suppuration. In both cases, the temperature rose to 105° F. every day with marked remissions to normal or below normal. There were no physical signs in the chest. Sinus thrombosis was suspected in both cases. After a week, a roentgen-ray picture of the chest was taken, and showed a lobar pneumonia.

Orbital cellulitis and frontal sinus disease may be mistaken for cavernous sinus thrombosis. The former is almost always unilateral, whereas cavernous sinus thrombosis remains unilateral for only a few days. In orbital cellulitis and frontal sinus disease there are no ocular paralyses, whereas in cavernous sinus thrombosis, paralyses of the ocular muscles and pupillary changes are common. Intranasal examination and roentgen-ray pictures often give some information in frontal sinus disease. The temperature is usually higher and more likely to be of the septic type in cavernous sinus thrombosis. There may be bacteriemia and metastases in the latter condition.

Acute follicular tonsillitis may be mistaken for sinus thrombosis, when the tonsillitis complicates a middle-ear suppuration. Very high temperature and bacteriemia may occur with tonsillitis. Throat symptoms frequently occur with sinus thrombosis, especially with bulb thrombosis, when there is involvement of the glossopharyngeal nerve, and in cavernous sinus thrombosis, when there is extension to the pterygoid plexus. It might be necessary to wait until the tonsillitis subsides, before making a diagnosis. Tobey's test might be of value.

Cervical adenitis occurring with middle-ear suppuration often confuses the clinical picture. This is especially apt to occur in children. Cervical adenitis often causes a temperature curve very much like that produced by otogenic sepsis, and simulates tenderness along the jugular. Occasionally cervical adenitis causes thrombosis of the internal jugular vein with extension to the sigmoid sinus. Although the temperature may be quite high with

cervical adenitis, the patient does not usually look or act as sick as with otogenic general sepsis. There is no bacteriemia in cervical adenitis.

Nasopharyngeal infections give rise to high temperatures, which may continue for several weeks and be confused with sinus thrombosis. The infection in the nasopharynx is usually the cause of the middle-ear suppuration. It is usually accompanied by cervical adenitis, and is much more common in children than in adults. Examination of the throat reveals a thick ropy mucopurulent secretion coming down from behind the soft palate.

Pyelitis is occasionally complicated by middle-ear suppuration. The temperature of a pyelitis is very much like that of otogenic sepsis. The condition can be readily diagnosed by examination of the urine. Metastases in the kidney may give rise to a similar picture.

The diagnosis of otogenic general sepsis is very easy in some cases and very difficult in others. In the pyemic cases, with marked remissions in the temperature, chills and metastases, the picture is typical. But there are a great many cases where the temperature is atypical, and where there are no metastases. In some cases the temperature is continuously high. This is especially likely to occur in the severe septic type. There are no chills and no metastases. A blood culture may help in these cases, but this is frequently negative. There are no localizing signs as there is usually no time for the formation of an obturating thrombus. There is either a mural thrombus or no thrombus at all. Examination of the sinus wall during the mastoid operation may give no clue. On incising the sinus, there is usually free bleeding. The diagnosis is made in these cases on the picture of a general sepsis occurring with a middle-ear suppuration, and from the fact that no other condition can be found to account for the severe general symptoms. If the condition lasts long enough, metastases may eventually appear.

Another class of cases in which the diagnosis is very difficult, is the group in which the general symptoms are very mild. These are the cases in

which the local picture predominates. The extreme cases of this type are the ones where the thrombus is found accidentally at operation. In the course of a mastoidectomy, a perisinous abscess is found. The sinus wall is covered with granulations; there is a fistula in the sinus wall, and a broken-down thrombus in the sinus, which has ruptured and is draining into the mastoid cavity. The ends of the thrombus are firm and not infected. Or else the sinus is accidentally uncovered during a mastoidectomy, and is found to be transformed into a firm solid cord. When the sinus is opened, it is found to be filled with an organizing or organized thrombus. In these cases a diagnosis is not important, because the patient gets well without additional operative intervention.

Holderman collected 31 cases of afebrile sinus thrombosis from the literature. Of these cases, 14 had an abscess within the sinus, with rupture through the outer sinus wall, and 17 had firm or totally organized thrombi. Cases of this type have been reported by Hoffmann, Warnecke, Uchermann, Grunert, Dallmann, Mahler and Day.

There is a large group of cases in which the septic symptoms are of moderate severity. The temperature does not go above 101° F. or 102° F. There are no sharp rises and falls, and no chills. There may be occasional slight chilly sensations. The patients are anemic and have pasty complexions. They may or may not have a bacteriemia. Metastases do not occur until late in the course of the disease. Griesinger's sign may be present; there may be tenderness along the course of the jugular; Tobey's sign may be positive; Crowe's sign may be positive. In many cases, the fact that the patient is not making the proper progress after a mastoid operation, and looks moderately septic, has headache, a persistent anemia and a persistent polynucleosis, makes the otologist suspect the possibility of a sinus thrombosis. He explores the sinus and finds a thrombus.

So-called primary sigmoid sinus thromboses are those in which the middle ear appears normal. What really happens in these cases, is that the middle-ear infection is so slight that no perforation of the drum-membrane occurs. The signs in the middle ear gradually disappear, while the infection in the mastoid and the sigmoid sinus persists. These cases are very difficult to diagnose, because they give no history of ear disease.

Primary bulb thrombosis is often impossible to diagnose without exploratory operation. There may be no mastoid involvement. There is an acute middle-ear suppuration with the immediate occurrence of septic symptoms. The mastoid is opened and found slightly or not at all involved. The sinus is incised and no thrombus is found. There is free bleeding from the upper end of the sinus, but no bleeding from the lower end. This makes the diagnosis of bulb thrombosis. Primary bulb thrombosis is most common in children, but it may occur in adults. In cavernous sinus thrombosis, the local signs are usually sufficiently well marked to make the diagnosis a simple matter.

A sinus thrombosis may run its course in three or four days and end fatally, or it may last for two or three months, and end in death or in recovery. Death may be due to:

1. Pyemic or general septic intoxication.
2. Extensive metastases in the lung or heart.
3. Septic endocarditis.
4. Meningitis or brain abscess.
5. Occasionally from sinus hemorrhage.
6. Occasionally from vagus paralysis.

Chapter V

TREATMENT

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TREATMENT

THE treatment of otogenic general sepsis cannot be schematized. A number of variable factors must be taken into consideration. The treatment will depend upon the severity of the inflammation of the sinus wall, the condition of the thrombus (whether organizing or broken down), the character and location of metastases, the character of the fever (presence or absence of chills, or the septicemic type of fever), and the general, cerebral and local symptoms. The cases with extensive local disease and mild general symptoms cannot be treated like the cases with slight or no local changes and severe general symptoms. Mild cases require no treatment other than removal of the original source of the infection, while severe cases will demand all of the otologist's resources and ingenuity, medical as well as surgical. However, the efforts of the otologist are often rewarded in a very gratifying way. Patients who appear desperately ill on one day, and who would undoubtedly die without the proper surgical interference, are often well on the road to recovery, a day or two after operation. The percentage of recoveries following operation for otogenic general sepsis is far greater than that following the other major intracranial complications of otitis, brain abscess and meningitis. The prognosis varies with the accessibility to surgical intervention of the sinus which is involved. The prognosis is best in thrombophlebitis of the sigmoid sinus, and is least favorable in thrombophlebitis of the cavernous sinus.

Percentage of Cures. A list of the percentage of cures of sigmoid sinus thrombosis in the hands of different operators is shown in Table 1.

The time at which operative interference is instituted influences the prognosis. The earlier the patient is operated on, the better is the prognosis.

TABLE I

Operator	Cases reported	Percentage of cures
Alexander	96	84
Körner	100	68
Mygind	80	56
Heine	68	47
Atkins	40	72.5
Rosenblatt	61	50
Knutson	55	78
Voss (Riga)	48	65
Hegener	48	58
Dench	44	77
E. Wolf	32	66
O. Voss	30	57
Bondy	30	60

Of 120 cases reported by Bezold, in which operation was done within a week of the onset of septic symptoms, 75 per cent recovered. Of 80 cases operated on after the first week, only 37.5 per cent recovered.

The virulence of the infecting organism and the resistance of the patient are important factors in the prognosis. The location of metastases is also important.

ORIGIN OF MODERN TREATMENT. The modern treatment of otogenic general sepsis dates back to 1880. In this year, at a meeting of the Verein der Deutschen Ärzte, in Prague, Zaufal advised ligation of the internal jugular vein and incision of the lateral sinus, with evacuation of any contained clot, for the treatment of otogenic general sepsis. He did not himself perform the operation until many years later. His suggestion was first followed by Trendelenburg in the treatment of thrombophlebitis of the pelvic veins in gynecologic conditions.

Lane performed the first operation on the lateral sinus and jugular vein in 1888. Ballance performed an operation on the sinus and jugular in 1889. He was the first to advise division of the jugular vein between two ligatures. Neither Lane nor Ballance knew of Zaufal's recommendation when they performed their first operations. In 1901, Viereck reported the results of 170 operations for sinus thrombosis. Of these cases 89 per cent recovered.

Indications for Treatment

The indications for treatment of otogenic general sepsis may be grouped under five heads:

1. To remove the original source of infection, that is, to perform a simple or radical mastoidectomy, with exposure of the sinus. In some mild cases of otogenic general sepsis, this suffices to cure the condition.

2. To evacuate the clot and remove as much of the infected sinus wall as is necessary to prevent spread of the infection in the sinus. (This refers also to the internal jugular vein, when the disease has spread to this vessel.)

3. To block the main venous channel through which the septic material is being poured into the general circulation, that is, ligation or resection of the internal jugular vein.

4. To increase the resistance of the body by improving the quality of the circulating blood, that is, the employment of blood transfusions. To this may be added the use of disinfecting materials, such as mercurochrome, intravenously.

5. To treat the metastases.

The second indication, the removal of the clot and the inflamed portion of the sinus wall, is met most easily when the sigmoid sinus is involved. It presents greater difficulty when the jugular bulb, the superior longitudinal

sinus or the superior petrosal sinus is involved. It presents the greatest difficulties when the cavernous sinus or the inferior petrosal sinus is the seat of the disease.

The simplest treatment for otogenic general sepsis is the performance of a mastoidectomy with exposure of the sigmoid sinus. In a case of acute middle-ear suppuration, in which, in spite of adequate drainage, high temperature persists after a number of days and especially if the temperature is of the remittent type with slight chilly sensations, a mastoidectomy should be done, and the sigmoid sinus should be uncovered at the knee. If the sinus wall looks normal, the blood culture is negative, and there are no other signs of general sepsis, nothing further should be done at the time. If a perisinous abscess is found, and the sinus wall is covered with granulations, nothing further need be done. In some of these cases the temperature drops after the sinus is uncovered and the patient goes on to recovery. We cannot be sure whether a thrombus is present in these cases or not. The sepsis may have been the result of absorption of septic material from the inflamed mastoid, or bacteria may have entered the circulation through the sinus wall. Thrombi sometimes organize when the source of infection is removed from around the sinus. If the sinus wall looks suspicious, or if the septic symptoms continue after the sinus has been exposed, the interior of the sinus should be examined. In order to make a satisfactory examination of the interior of the sinus, bleeding must be completely controlled. This is done by inserting two plugs of iodoform gauze between the outer sinus wall and the overlying bone, one above and one below the point which is being examined. This method of controlling sinus bleeding was first described by Whiting in 1899. It was described by E. Meier, independently, the following year. The sinus should be uncovered for at least 1 inch, from the knee downward.

The initial exposure of the sinus should be made with a gouge that has a deep shoulder (Fig. 83). The gouge should be placed over the sinus plate

at the knee, almost parallel to the surface of the sinus. The shoulder of the gouge prevents injury to the sinus wall. A broad curette may be used instead of the gouge. After a small portion of the sinus is uncovered, the exposure is

FIG. 83. REMOVING INNER TABLE OVER SIGMOID SINUS WITH GOUGE.

completed with a broad-tipped rongeur, a small portion of the sinus plate being removed at a time (Fig. 84). One blade of the rongeur is introduced between the sinus wall and the overlying bone, carefully separating the sinus from the bone, and the rongeur is withdrawn about $\frac{1}{16}$ inch before the fragment of bone is cut through, in order to avoid pinching the sinus wall between the blades of the rongeur. In this way it is almost impossible to injure the sinus wall. Richards advises the use of a gouge or chisel for the entire exposure of the sinus. He believes that there is less likelihood of breaking off particles of thrombus in this way, than by using a rongeur, as the latter exerts a certain amount of pressure on the contents of the sinus.

The exposure must include the entire width of the sinus, and should extend a little beyond the border of the sinus on either side. The tampon should consist of a tightly rolled piece of iodoform gauze, about ½ inch

FIG. 84. ENLARGING OPENING IN INNER TABLE OVER SIGMOID SINUS WITH RONGEUR.

thick, and a little wider than the sinus. If the tampon does not cover the entire width of the sinus, bleeding will not be completely controlled. The upper tampon should not be placed above the knee of the sinus, for if the superior petrosal sinus, which enters the sigmoid sinus at the anterior margin of the knee, is not thrombosed, it would give rise to annoying bleeding, which would interfere with examination of the interior of the sinus.

When both tampons are in place, a vertical incision is made in the outer sinus wall, from ½ to ¾ inch in length, being careful not to injure the visceral

wall of the sinus (Fig. 85). A very sharp knife should be used, and the belly of the blade should be employed. The outer sinus wall may be raised with mouse-toothed forceps, in order to facilitate the incision. The lips of

FIG. 85. INNER TABLE REMOVED OVER KNEE OF SINUS. LUMEN OF SINUS OCCLUDED ABOVE AND BELOW BY TAMPONS. OUTER WALL OF SINUS INCISED.

the wound are then separated with forceps, and the interior of the sinus carefully examined. In this way, even a small mural thrombus can be discovered.

If the sinus contains no clot at this point, the upper plug is removed. If there is free bleeding from above after removal of the plug, there is probably no thrombus in the sinus above the point which is being examined. This conclusion is only probable, because the bleeding may come from the superior petrosal sinus. It is very unlikely, however, for a thrombus to be

present above the knee of the sinus, except as an extension of a thrombus at or below the knee. The only way to be sure that the bleeding does not come from the superior petrosal sinus is to uncover the sigmoid sinus further up, plug it off well above the knee, extend the incision in the sinus wall above the point of entrance of the superior petrosal sinus, and then observe the point of entrance of the superior petrosal by direct vision. Another method of determining whether the superior petrosal is thrombosed is to block off the sigmoid sinus above and below the knee, and incise the sinus wall between the two plugs. If there is bleeding, the superior petrosal sinus is patent.

If it is determined that there is no thrombus above the knee of the sinus, the upper plug is replaced and the lower plug is removed. If there is no bleeding from below, there is a thrombus in the lower part of the sigmoid sinus, in the jugular bulb, or in the internal jugular vein. If there is a small amount of bleeding from below, there is probably a mural thrombus in the lower part of the sinus or the bulb. If there is free bleeding from below, there is probably no thrombus in the lower part of the sigmoid sinus or in the bulb. It is possible, however, for a thrombus to be present in the lower part of the bulb, with free bleeding from the lower end of the sinus. The bleeding may come from the inferior petrosal sinus, or the condyloid emissary, or from both of these vessels.

A simple method of demonstrating how much of the bleeding from the lower end of the sinus comes from the inferior petrosal sinus, and how little of it comes from the internal jugular vein, is to remove the lower plug and then compress the internal jugular vein in the neck. Instead of checking the bleeding from the lower end of the sinus, this manipulation will increase the bleeding. This shows conclusively that most of the bleeding is from the tributaries to the bulb, which, having their usual egress through the internal jugular vein blocked, are now diverted upward through the incision in the sinus wall. When pressure on the internal jugular vein increases the bleeding

from the lower end of the sinus, we are sure that there is no obturating thrombus in the bulb.

Richards advises placing the plugs lightly over the upper and lower ends of the sinus before incising the sinus wall, and not obliterating the sinus by pushing the plugs between the sinus wall and the overlying bone. If, on incising the sinus wall, there is free bleeding, the upper end of the sinus should be compressed by the plug. If there is still a free flow, the lower end of the sinus should be compressed. By opening the sinus before applying pressure, he believes that an opportunity is given for the escape of pieces of clot which may have been loosened by manipulation. The pressure plugs are likely to cause pieces of the clot to break off, if the thrombus extends beyond the point which is compressed.

If a thrombus is found in the sigmoid sinus, the internal jugular vein should be tied off before anything further is done to the sinus, in order to prevent particles of thrombus from being carried through the jugular vein into the general circulation. Tying off the jugular also prevents the occurrence of air-embolism. The operation on the sinus is then completed. The incision in the sinus wall is extended with scissors above and below, until the end of the thrombus is reached (Fig. 86). The thrombus is lifted out with forceps. No attempt should be made to curette the interior of the sinus through a small opening in the sinus wall. Such an attempt might cause injury to the visceral wall of the sinus. One cannot be sure that the entire thrombus has been removed, unless it is done under direct vision. Furthermore, even if the entire thrombus is removed, the inflamed sinus wall remains. This is apt to give rise to another thrombus. If the thrombus extends beyond the limits of the original sinus exposure, the exposure must be extended. It is continued in an upward and backward direction, until the outer sinus wall looks normal. Another compression plug is placed over the sinus, and the incision in the sinus wall extended as far back as possible. If the end of the thrombus

is not reached, this procedure should be repeated, until the end of the thrombus is reached. Bleeding from the upper end of the sinus is controlled by a packing placed between the outer sinus wall and the overlying bone,

FIG. 86. SINUS OCCLUDED ABOVE AND BELOW. OUTER SINUS WALL INCISED AND CLOT REMOVED.

and not into the interior of the sinus. If the latter method is employed, there will be free bleeding when the packing is removed several days later, as the lumen of the sinus is kept open. When the packing is placed between the outer sinus wall and the overlying bone, however, the lumen of the sinus is obliterated, and adhesions occur between the outer and inner sinus walls. These adhesions become very firm in a few days, and there is no bleeding when the packing is removed.

The same procedure is now followed at the lower end of the sinus. The sinus is exposed as far down as possible toward the bulb. It is possible to

reach almost to the bulb, by continuing the bone removal from the region of the mastoid tip, through the basilar portion of the occipital bone. When the sinus is exposed as far down as possible, a compression plug is placed over

FIG. 87. OUTER WALL OF SINUS REMOVED.

the lower end of the sinus, and the sinus wall split up to the plug. If it is possible to reach the lower end of the thrombus, the thrombus is lifted out with forceps. The entire outer wall of the sinus is then removed with scissors, and the sinus is converted into an open trough (Fig. 87). The interior of the sinus becomes part of the open mastoid cavity. If the lower end of the thrombus cannot be reached in this way, the bulb may have to be opened. This procedure will be described later.

After the thrombus has been completely removed, and the outer sinus wall resected, the cavity is loosely packed with iodoform gauze, and the skin incision partly sutured.

FIG. 88. SINUS OPENED AS FAR BACK AS TORCULAR.

Voss, Brieger and other otologists advise leaving the ends of the thrombus in situ, if they seem firm, and removing only the central broken-down portion of the thrombus. They believe that the firm ends of the thrombus act as a protection against the spread of the infection. However, it is impossible to tell whether the ends of the thrombus are infected or not. A thrombus may look firm and uninfected, and may nevertheless contain many bacteria. Bacteria may be washed into the general circulation from these apparently benign portions of the thrombus. It is far safer to remove

the entire thrombus. Furthermore, the sinus wall is usually inflamed as far as the thrombus extends, and if the ends of the thrombus are left in situ, the inflammation in the sinus wall may continue to extend, and cause an extension of the thrombotic process.

If, in the presence of a septic temperature, the outer sinus wall is found to be discolored and show evidence of necrosis, and there is no thrombus in the sinus, the sinus should be blocked off above and below the diseased area, and the outer sinus wall should be resected; for such a diseased sinus wall may be the source of a general sepsis, and it is likely that a thrombus will form sooner or later, if the sinus is left alone.

If, after removal of the thrombus as described above, the septic symptoms continue, the wound must be reopened, and the interior of the sinus examined above and below the limits of the first operation. If the thrombotic process has extended, it must be followed up to the limit. It is sometimes necessary to go as far as the torcular (Fig. 88), or even beyond the torcular into the superior longitudinal sinus or the opposite lateral sinus.

Jugular Ligation

There is a diversity of opinion as to the advisability and the value of jugular ligation in the treatment of otogenic general sepsis. Some otologists advise jugular ligation in every case of otogenic general sepsis. Others advise that it should never be employed. The majority advise its use only in selected cases.

Numerous statistics have been collected to show the value or the uselessness of jugular ligation. Körner has collected statistics which are shown in Table II.

According to these statistics, the results were about the same with as without jugular ligation.

TABLE II

Operative method	Number of cases	Doubtful result	Death	Cure
Jugular ligation without opening sinus	9	0	4	5
Opening sinus without jugular ligation	132	0	55	77
Jugular ligation before opening sinus	94	2	36	56
Jugular ligation after opening sinus	68	1	29	38
Time of opening sinus and jugular ligation not given	5	0	1	4
Total	308	3	125	180

Knutson reported the results in 55 cases operated upon by himself. The jugular was ligated in 22 cases, with 5 deaths (22.7 per cent). The jugular was not ligated in 33 cases, with 7 deaths (21.3 per cent).

Grünberg collected statistics which are shown in Table III.

TABLE III

Authors	With ligation of vein		Without ligation of vein	
	No. of cases	Per cent of cases	No. of cases	Per cent of cases
Rodenberg	27	85	0	0
Reik	24	75	42	43
Wesselowsoroff	18	67	9	29
Jansen	12	70	10	100
Jansen	20	65	30	73
Abrashanoff	125	64	100	51
Körner	94	60	132	58
Körner	68	56	0	0
Heine	14	43	60	50
Halle	12	25	56	52
Botey	4	0	4	100

The percentage of cures in the hands of various operators with ligation of the vein is from 0 per cent to 85 per cent, and without ligation of the vein from 29 per cent to 100 per cent. No definite conclusions can be drawn from these statistics. They are of no value because no attention is paid to the different malignity in different cases, and to indications for ligation in various cases. Some surgeons employ ligation only in very serious cases, and consequently have a much higher mortality rate in the ligated cases than in the non-ligated cases.

Objections to Jugular Ligation. There are three objections which are usually brought forward against jugular ligation:

1. Ligation of the jugular does not close off every avenue for the spread of infection from the thrombotic area.

2. In a few cases, it has proved dangerous, on account of subsequent circulatory disturbances in the brain.

3. Ligation of the jugular may cause spread of the thrombus into the inferior petrosal and cavernous sinuses.

With regard to the *first objection*, it is true that the internal jugular vein is not the only pathway for the dissemination of infectious material from the sigmoid sinus. There are four other pathways through which the infectious material may enter the circulation. They are:

1. Through the inferior petrosal sinus to the cavernous sinus, then to the facial, external jugular and subclavian veins. Or, from the petrosal sinus to the cavernous, and then through the circular sinus to the opposite cavernous and the opposite internal jugular.

2. Through the condyloid emissary to the spinal plexus, the vertebral plexus and the subclavian vein.

3. Through the marginal sinus to the occipital sinus, the opposite marginal sinus and the lateral sinus and jugular vein of the opposite side.

4. Through the mastoid emissary to the occipital vein and the subclavian vein.

Besides these pathways, infection can reach the blood stream through the lymph vessels which empty into the junction of the subclavian and internal jugular veins. The infectious material may also travel from the thrombus through the vasa vasorum into the general circulation.

To this objection, it may be answered that even though the jugular vein is not the only pathway for the spread of the infectious material into the general circulation, it is by far the most important pathway, and it is an advantage to block it off. Furthermore, when the thrombus involves the lower end of the sigmoid sinus and the bulb, the thrombotic material may block the mouths of the inferior petrosal sinus, the condyloid emissary and the marginal sinus. If this lower portion of the thrombus is not infected, there will be no extension along these tributary pathways. When the sinus and bulb are opened, and free drainage is established from the interior of the sinus and bulb, there is still less tendency to extension through the tributaries. The mastoid emissary is blocked in the operation on the sigmoid sinus, thus minimizing the danger of extension through this pathway.

Furthermore, the prevention of spread of the infection is not the only purpose in ligating the jugular vein. Its purpose is also to set the lower limit to the portion of the venous system which is cut out of the circulation by the sinus operation. The upper limit of the area which is cut out of the circulation is fixed by the plug which compresses the upper end of the lateral sinus. As it is frequently impossible to reach the lower limit of the thrombus in the sinus, the lower limit of the area to be cut out of the circulation must be set by the ligation of the jugular. Ligation of the jugular is always combined with a wide opening of the entire ligated venous channel up to the point of infection.

Ligation of the jugular preliminary to the sinus operation also minimizes the danger of pieces of thrombi being carried into the circulation from manipulations on the sinus. It also prevents air-embolism.

The *second objection* made to jugular ligation is the danger of circulatory disturbances in the brain, when the opposite jugular is very small. Rohrbach, Linser, Kummer and Stolz each reported a case in which death followed jugular ligation. In each one of these cases, there was incomplete development of the opposite lateral sinus and jugular. In the case reported by Linser, the jugular foramen on the affected side was 11 × 14 mm. and on the opposite side, 5 × 6 mm. Occasionally there are temporary symptoms of circulatory disturbance, such as edema of the face, cyanosis and unconsciousness, lasting for a short time. Linser showed that in 1000 skulls, one jugular foramen was absolutely contracted (3 to 4 mm.) in 3 per cent of the cases.

If there is an obturating thrombus in the sinus, ligation does not add to the circulatory embarrassment. Circulatory disturbances are rare with obturating thrombi because the formation of the thrombus is so slow that there is time for the establishment of collateral circulation. If there is a parietal thrombus or no thrombus at all, it might be possible to prevent unpleasant results by ascertaining the sufficiency of the opposite jugular by the method of Crowe, before attempting ligation. If there is dilatation of the retinal and supraorbital veins on compression of the jugular vein on the affected side, the opposite jugular is insufficient to carry on the circulation. In these cases, the jugular should not be tied.

Several cases have been reported in which it was necessary to ligate both internal jugular veins, and the patients recovered. In these cases the collateral circulation through the emissaries and through the canals of Breschet, and the communications with the venous network in the spinal canal, were sufficient to keep up the circulation in the skull. Hastings reported a case of double jugular resection, the two operations being done

seventeen days apart. The patient recovered. Baldwin reported a case in which both sides were operated on simultaneously. The patient recovered. Ewald reported a case with double ligation, in which there was immediate death. Dangel reported a case in which there was recovery from the operation, but in which death occurred four weeks later from erysipelas. Duval reported a case with recovery. Recovery following resection of one jugular with bilateral sinus operation was reported in 6 cases.

As a matter of fact, the danger from circulatory disturbances in jugular ligation is so remote that it does not weigh against the advantages of this operation.

The *third objection* to jugular ligation is the likelihood of extension of the thrombus into the inferior petrosal sinus and cavernous sinus. It is contended, that when the jugular is ligated, there is stasis of the blood column from the ligated point up to the jugular bulb. This column of blood clots, and may become infected, and the thrombus then extend into the inferior petrosal and cavernous sinuses. This contention was not supported by Haymann's experiments. Haymann found on autopsy that in the dogs in which he infected the lateral sinus and ligated the jugular vein, the vein above the ligature was collapsed, and did not contain a thrombus.

In 47 cases of sinus thrombosis examined at autopsy by Török, jugular ligation had been done in 31. Of these 31, there was thrombosis of the inferior petrosal sinus in 7. In 16 without jugular ligation, 4 had thrombosis of the inferior petrosal. This shows that jugular ligation does not favor the formation of a thrombus in the inferior petrosal sinus.

As the jugular ligation is always followed or preceded by opening of the sinus, the danger of retrograde extension is minimized, even if a clot should form above the ligated point.

The only cases of obturating thrombosis in which jugular ligation is not necessary are those in which the thrombosis is definitely localized about

the knee or above the knee of the sinus. In these cases the thrombus is easily removed from the sinus in toto, and the lower part of the sinus is normal.

When the thrombus extends down into the lower part of the sinus, or into the bulb or the internal jugular vein, the jugular must be tied off before any manipulations are undertaken on the sinus.

When there is a parietal thrombus in the bulb or jugular vein, the vein must be ligated. When there is a parietal thrombus in the upper part of the sinus, there is no need of ligating the jugular unless there are very severe signs of sepsis, or unless there are metastases.

If there is a phlebitis of the sinus without thrombus formation, and only moderate signs of sepsis, the vein need not be ligated. If there is phlebitis of the sinus with severe septic signs or with metastases, the vein must be ligated.

In every case of otogenic general sepsis with metastases, the vein should be ligated.

If the sinus is operated on, and the jugular is not ligated, and the septic symptoms persist for several days, the vein should be ligated at a secondary operation.

If a thrombus is found in the sinus, and there are no signs of sepsis, that is, if there is no fever and there are no metastases, the jugular need not be ligated.

The jugular should never be ligated until the sinus has been exposed and examined, except in cases where it is possible to make a positive diagnosis of obturating thrombus before the mastoid operation has been done. Such cases are rare, except when metastases have already occurred.

If the thrombus does not extend into the jugular vein, the latter should be ligated as high up as possible, so that if the bulb is involved, drainage from the latter will have as short a distance as possible to travel. The ligation should be done above the entrance of the facial vein into the jugular,

as the former communicates freely with the external jugular. If ligation is done below the facial, the infectious material in the blood would travel from the internal jugular through the facial to the external jugular. Ligation above the facial also allows the facial vein to aid in establishing collateral circulation.

Jugular ligation should never be done without operation on the sinus.

There is no unanimity of opinion as to the relative values of ligation and resection of the internal jugular vein. Tobey advises ligation rather than resection of the vein. In over 200 cases operated upon at the Massachusetts Charitable Eye and Ear Infirmary, it was found necessary to resect the vein in only 2 cases. In these 2 cases there was infection of the jugular below the facial vein. The advantages of ligation over resection are, the shorter time required for the performance of the former operation, the small amount of shock, the non-exposure of the tissues of the neck to infection, and the small insignificant scar.

There is no advantage in dividing the vein between the two ligatures. For if there is a bacteriemia, division of the vein is likely to cause an infection of the tissues of the neck about the point of ligation. The purpose of the division of the vein is to prevent spread of a phlebitis of the wall of the vein beyond the point of ligation.

Resection of the jugular vein is necessary only if there is a thrombus in the vein itself, or if there is a phlebitis and periphlebitis of the vein. In such cases, ligation is of no value.

Technique for Jugular Ligation. The incision for jugular ligation may be either vertical or horizontal. The horizontal incision gives a better cosmetic result, as it may be done in one of the natural creases of the neck. The vertical incision gives a little better exposure of the operative field.

In performing a jugular ligation, the patient's neck should be placed on the stretch. The patient should lie flat on his back, with his head turned

to one side. A folded sheet is placed under the shoulders and neck, so that the head falls back. This stretches the neck.

If a vertical incision is used, it should be made along the anterior

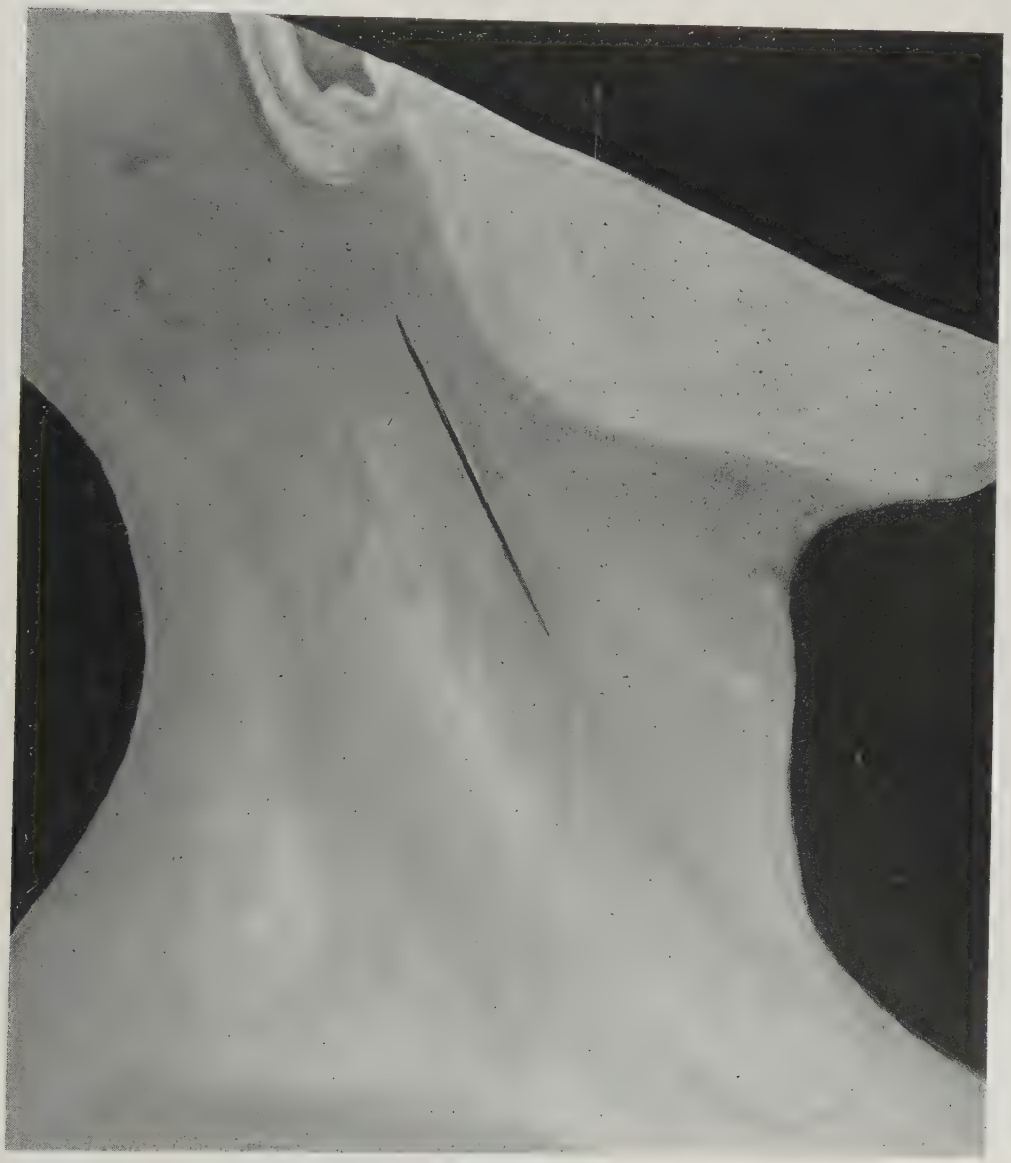

FIG. 89. SKIN INCISION FOR LIGATION OF INTERNAL JUGULAR VEIN.

border of the sternomastoid (Fig. 89). It begins about 1 inch above the angle of the jaw and extends downward for a distance of 2 to 3 inches. If a horizontal incision is used, it is made in one of the natural transverse folds

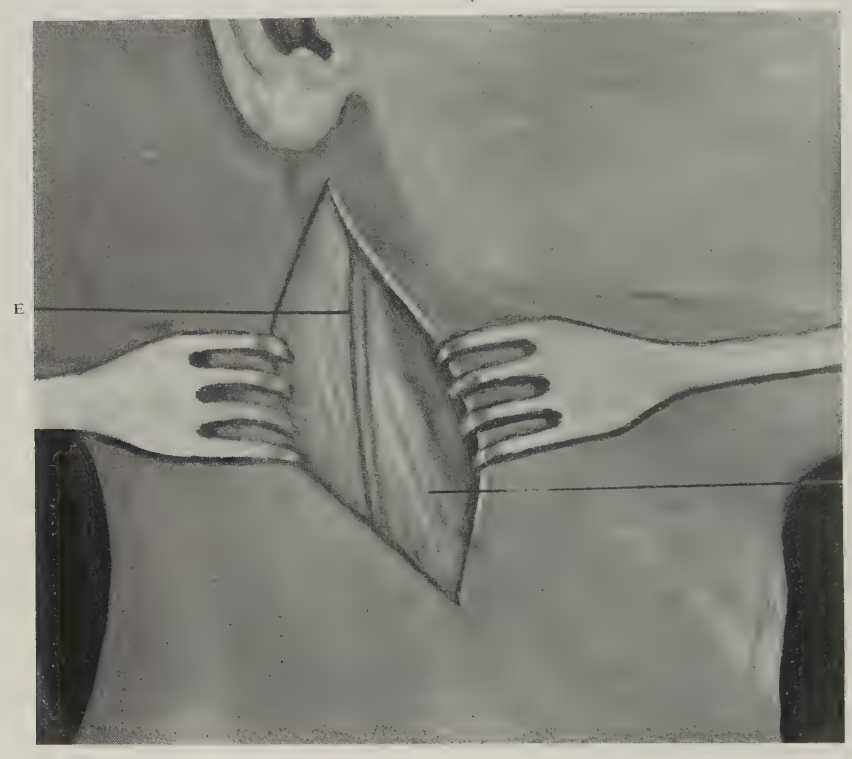

FIG. 90. JUGULAR LIGATION. SKIN, SUPERFICIAL FASCIA AND PLATYSMA DIVIDED.

E, external jugular vein; S, sternomastoid muscle.

of the skin, level with the upper margin of the thyroid cartilage. It should be about 1½ inches long. The incision is made through the skin, the superficial fascia and the platysma (Fig. 90). The external jugular vein should not be divided, but should be pushed back out of the way. The superficial layer of the deep fascia is then incised along the anterior border of the sternomastoid muscle. Avoid the anterior jugular vein, which lies a short distance in front of the sternomastoid muscle. Care must be taken to make

the incision through the fascia along the anterior border of the sternomastoid muscle, and not through the muscle, as in the latter case there is greater difficulty in finding the vessels.

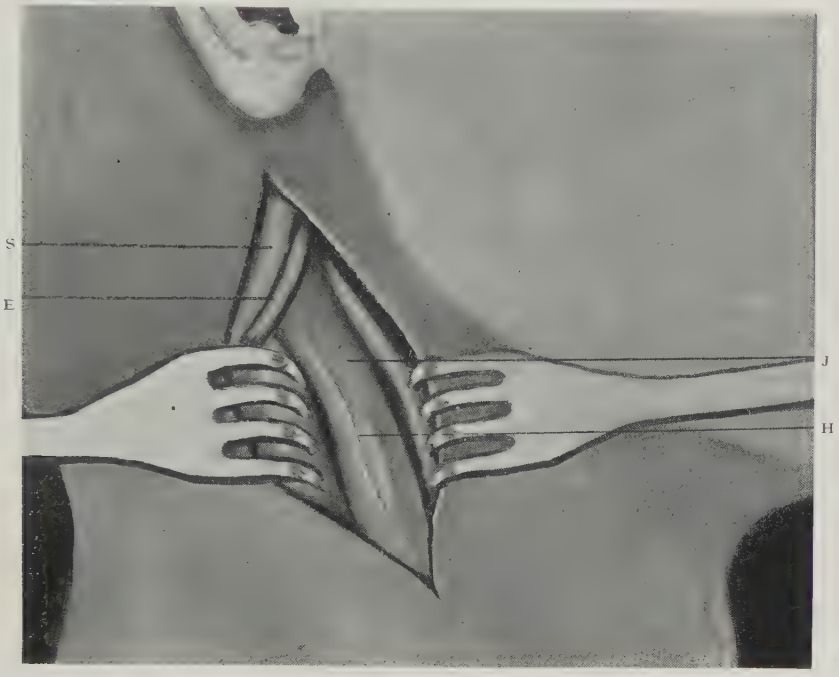

FIG. 91. JUGULAR LIGATION. STERNOMASTOID MUSCLE RETRACTED, EXPOSING SHEATH OF VESSELS.

E, external jugular vein; H, nervus communicans hypoglossi; J, sheath of great vessels; S, sternomastoid muscle

The sternomastoid is now retracted backward, and after a little blunt dissection, the sheath of the great vessels is brought into view (Fig. 91). The sheath of the vessels is formed by a portion of the middle layer of the deep cervical fascia. The vein is superficial and slightly posterior to the artery. The pneumogastric nerve is behind and between the artery and vein.

The jugular vein is seen before the carotid artery comes into view. The vein is easily recognized by its blue color and thin wall, and by the fact that it empties with each inspiration and refills with each ex-spiration. The

position of the carotid artery can be readily determined by feeling its pulsation. The sheath of the vessels is opened by pulling it away from the vessels with mouse-toothed forceps, and making a small incision in the sheath

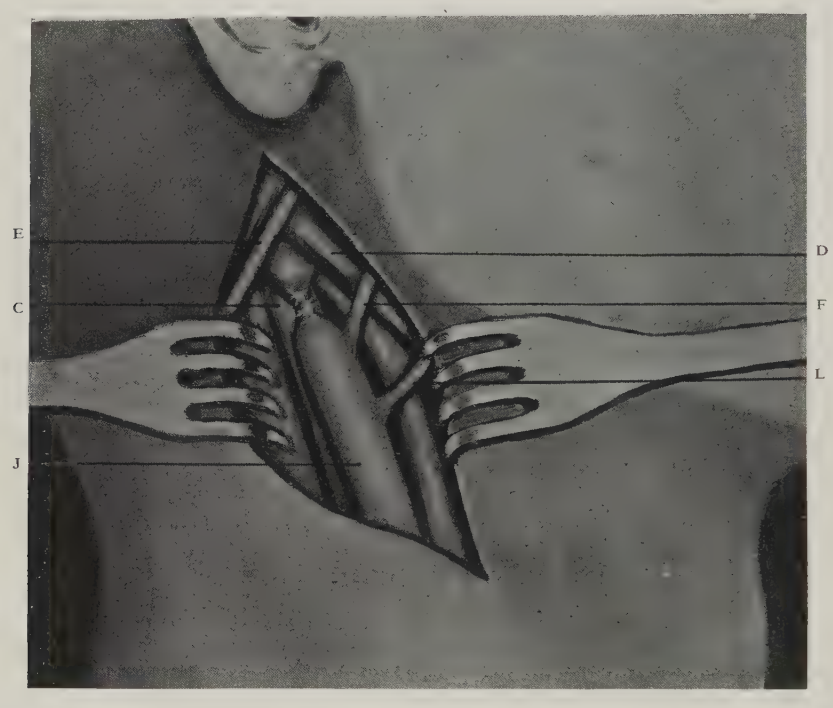

FIG. 92. SHEATH OF GREAT VESSELS DIVIDED, INTERNAL JUGULAR VEIN SEPARATED FROM SURROUNDING TISSUES, AND DOUBLE LIGATED ABOVE FACIAL VEIN.

C, carotid artery; D, anterior belly of the digastric muscle; E, external jugular vein; F, facial vein; J, internal jugular vein; L, lingual vein.

with the point of a scalpel, and then enlarging the opening with blunt dissection. The jugular vein is then carefully separated from the carotid artery and pneumogastric nerve by blunt dissection. The facial vein is identified, and the point of entrance of the facial into the internal jugular vein is noted. A double ligature of No. 1 chromic catgut is passed around the jugular above the point of entrance of the facial. Care must be taken not to include the pneumogastric nerve. The upper ligature is forced upward for about 1 inch, separating the vein from the artery as it is pulled upward.

By the use of traction on the ligatures, the lumen of the vein may be alternately opened and closed, and the patency or obstruction of the vein may be readily determined. If the vein is found to be patent, the ligatures are tied. If a thrombus is found in the vein, or if there are periphlebitic changes, the vein is resected. If a simple ligation is decided on, the upper ligature is tied first. The blood is expressed from the vein between the two ligatures, and the lower ligature is then tied (Fig. 92).

All bleeding points are carefully tied, and the fascia and skin sutured. A small cigarette drain is placed in the lower end of the wound. This is removed in two or three days, and if there is no secretion in the wound, it is not replaced. If the wound becomes infected, the sutures are removed, and the wound is packed.

If it is decided to resect the vein, the skin incision should be extended upward to within 1 inch of the lower extremity of the mastoid incision, and downward to ½ inch above the clavicle (Fig. 93). The incision in the deep fascia along the anterior border of the sternomastoid is extended downward as far as necessary (Fig. 94). The sternomastoid is retracted. This brings into view the omohyoid muscle, crossing obliquely over the lower part of the internal jugular vein (Fig. 95). The omohyoid muscle is divided. The vein is now separated by blunt dissection from the carotid artery and pneumogastric nerve, along its entire length. Inflamed cervical glands around the vein are not removed unless they are broken down. The facial, lingual and thyroid veins are isolated, double-ligated, and divided between the ligatures. The jugular vein is now divided between the two ligatures at its upper end. If the vein is not completely separated from the surrounding tissues, this separation is now completed. The vein is double-ligated at its lower end, the operator being sure that the ligatures are placed below the lower end of the thrombus in the vein. The vein is divided between these two ligatures (Fig. 96). The upper end of the vein is now dissected up toward the base of the skull as far

as possible. If the upper end of the vein contains a thrombus, or is empty, it is slit open, to provide drainage from the bulb. Alexander sutures the opened upper end of the vein into the skin, making a jugular-skin fistula. The neck

FIG. 93. INCISION FOR JUGULAR RESECTION.

wound is then packed, and the upper part of the wound sutured. It is unwise to sew up the neck wound completely, as there is always a certain amount of infection from the inflamed vein and the tissues about the vein.

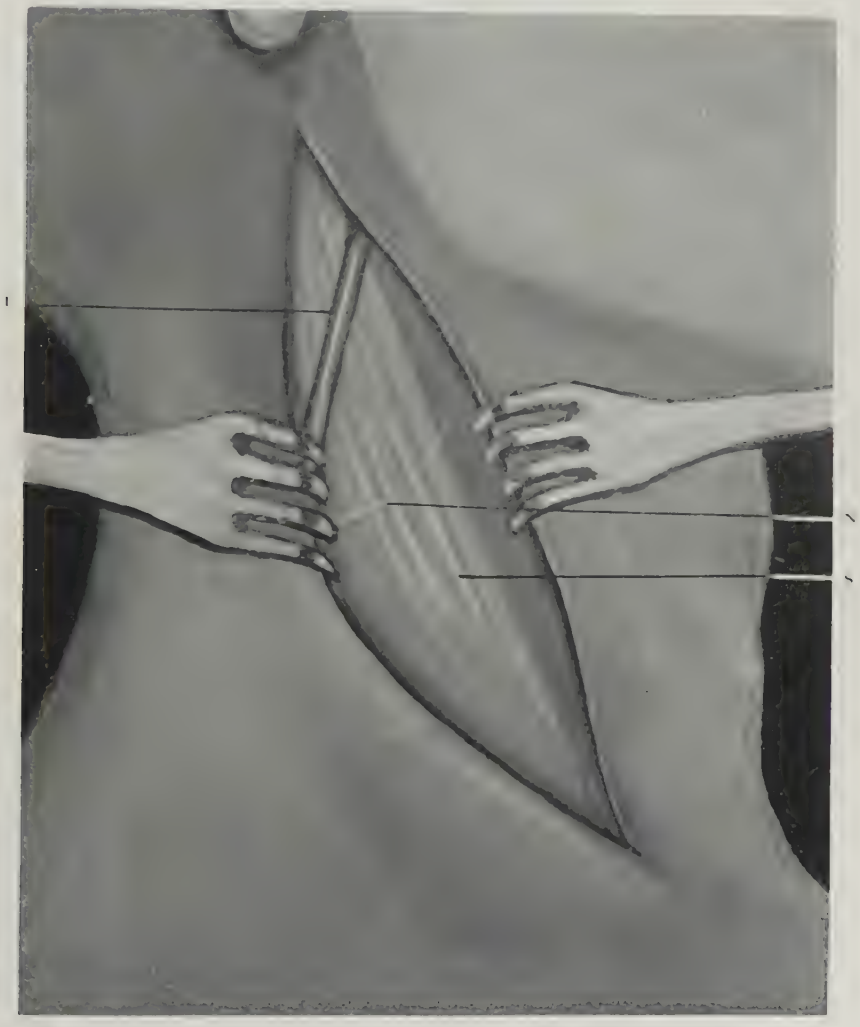

FIG. 94. JUGULAR RESECTION. SKIN, SUPERFICIAL FASCIA AND PLATYSMA DIVIDED AND RETRACTED.

E, external jugular vein; N, superficial cervical nerve; S, sternomastoid muscle.

Alexander describes his procedure as follows:

"If there is no blood in the distal portion of the vein, it is divided between the two ligatures, and the upper ligature is removed. The opening

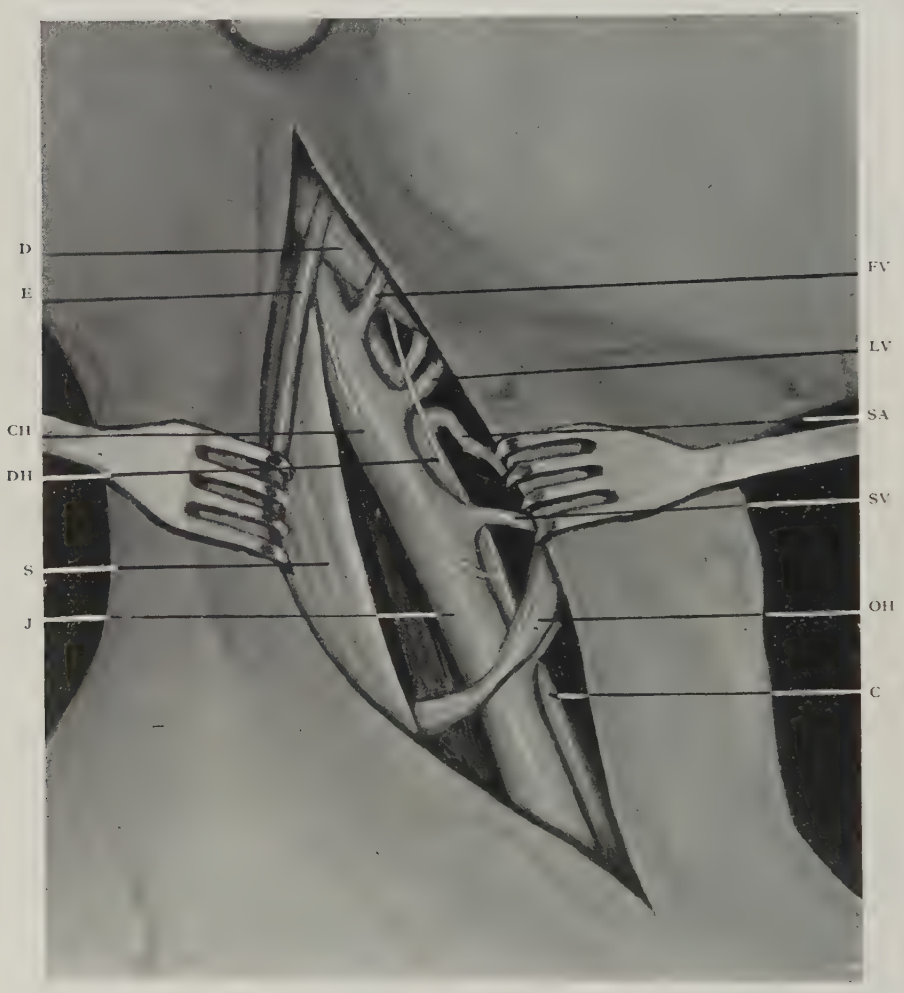

FIG. 95. JUGULAR RESECTION. SHEATH OF VESSELS OPENED AND INTERNAL JUGULAR VEIN FREED FROM SURROUNDING TISSUES.

C, carotid artery; CH, nervus collunicana hypoglossi; D, digastric muscle; E, external jugular vein; FV, facial vein; J, internal jugular vein; LV, lingual vein; OH, omohyoid muscle; S, sternomastoid muscle; SA, superior thyroid artery; SV, superior thyroid vein.

in the upper end of the vein is enlarged by a vertical incision with scissors. This results in an oval opening of the end of the vein. The cut edges of the vein are sewed into the upper end of the skin incision with a row of sutures.

FIG. 96. JUGULAR RESECTION COMPLETED.

CC, common carotid artery; E, external jugular vein; EC, external carotid artery at point where it gives off lingual and superior thyroid branches; FV, facial vein ligated; H, hypoglossal nerve; IC, internal carotid artery; LJ, lower stump of internal jugular vein; LV, lingual vein ligated; OH, omohyoid muscle divided; P, pneumogastric nerve; ST, superior thyroid vein ligated; UJ, upper stump of internal jugular vein.

The skin wound is then made smaller by sutures or Michel clamps, and a drain placed in the lower end of the wound [Fig. 97]. A probe can be passed

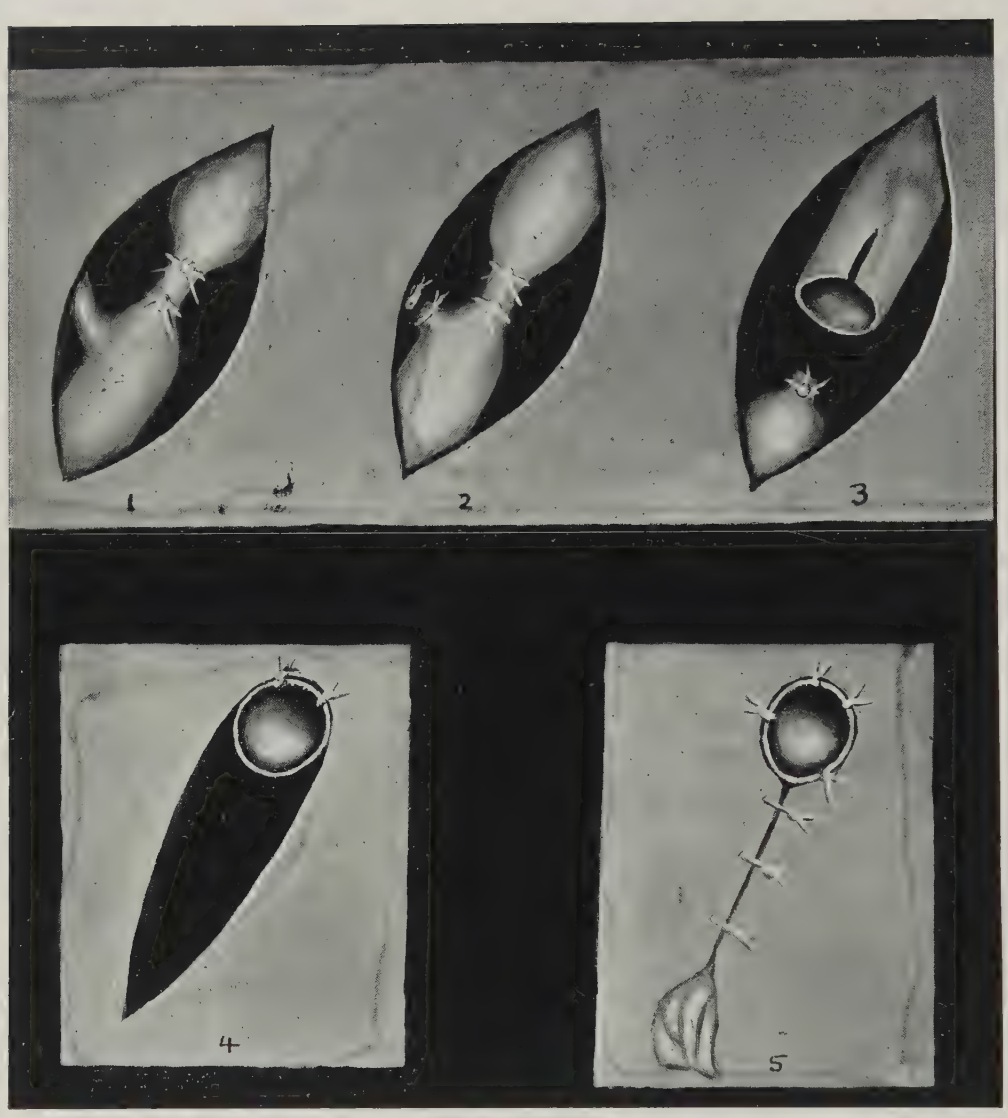

FIG. 97. JUGULAR SKIN FISTULA OPERATION. (*After Alexander.*)

1. Internal jugular vein double-ligated above facial vein.
2. Facial vein double ligated and divided between ligatures.
3. Internal jugular vein divided between ligatures, and upper end of vein partly split up.
4. Upper end of vein sewed into skin wound.
5. Skin wound sewed, with drain in low end of wound.

from the opening in the vein directly into the bulb. The vein becomes a natural drainage tube for the lower end of the sinus, bulb and upper part of the jugular vein. If there is blood in the vein, the upper ligature is left in situ. The upper end of the jugular is then fixed by sutures to the skin wound. Two to four days later, the ligature is removed. The skin wound can then be partly sutured. The secretion from the vein gradually lessens, and the vein obliterates in from one to three weeks."

If the septic temperature continues after operation on the sinus and ligation of the jugular, it is probably due to one or more of the following causes:

1. The thrombus may have extended toward the torcular. This can be determined by a revision of the wound. The phlebitis in the sinus frequently extends beyond the thrombus, so that even if the thrombus has been completely evacuated at the primary operation, there may be an extension of the thrombotic process after the operation. This extension must be followed up to the limit. Occasionally it is necessary to revise the wound a third time. It may be even necessary to follow the process into the superior longitudinal sinus or into the opposite lateral sinus.

2. The thrombus may have extended into the jugular bulb, or into the internal jugular vein. A parietal thrombus may have been present in the bulb at the primary operation. This cannot always be diagnosed. It may become obturating after the operation. When the thrombus extends into the bulb, it is usually necessary to open the bulb.

3. There may be an infection of the neck wound. Removal of the packing from the neck wound two or three days after operation is frequently followed by the evacuation of a large amount of pus. If the skin wound was sutured, the sutures should be removed. Infection of the neck wound may give a temperature of the same type as that caused by sinus thrombosis.

4. There may be erysipelas of the mastoid wound or neck wound. When the redness in the skin appears early, the diagnosis is easy.

5. The persistence of temperature may be due to the occurrence of metastases which started before the operation on the sinus and jugular. As several days usually elapse before the metastases become manifest, we must not assume that because a metastasis appears a few days after operation, the operation did not completely eradicate the source of infection in the sinus.

6. There may be an intercurrent acute infection, which has no connection with the sepsis, such as acute tonsillitis or pneumonia.

7. There may be an infection of the meninges or brain, due to extension from the wall of the sinus.

8. There may be thrombosis of one of the other sinuses. Occasionally there is a thrombosis of the opposite lateral sinus, which is not an extension from the original thrombus, but which is due to a mastoiditis of the opposite side.

9. The persistence of temperature is due, rarely, to iodoform poisoning. As soon as the iodoform packing is removed, the temperature drops.

It is not necessary to open the jugular bulb in every case of bulb thrombosis. If, after opening the sigmoid sinus, the thrombus in the sinus is found to extend down into the bulb, so that, after removal of as much of the thrombus as is accessible from the sinus, no bleeding is obtained from below, the sinus should be exposed as far down as possible through the mastoid wound. It is possible to reach almost to the bulb in this way. Even if pus wells up from the region of the bulb, it is unnecessary to open the bulb at this time. The jugular vein is tied off, and developments are awaited. Not infrequently, the temperature drops after this procedure. If the temperature stays down, nothing further need be done. If the jugular vein contains blood at the time of ligation, it should be left alone. But if, at a later dressing, it is found that the vein is thrombosed, it should be incised. Irrigation of the bulb from above or below is unsafe, as infectious material may be forced into the

inferior petrosal. A gauze drain may be passed into the bulb from above, and if any pus is present in the bulb, it may be carefully wiped out with an applicator.

In primary bulb thrombosis, a similar course is pursued. If the septic temperature persists for several days after the sinus is opened as far down as possible, it is necessary to attack the bulb operatively.

When, at the primary operation on the sinus, it is found that the bone disease extends to the jugular foramen, and there is a peribulbar abscess, an immediate operation must be done on the jugular bulb.

Technique for Operations on Bulb

There are two methods of attacking the bulb. In one method, the sinus is followed through the mastoid down to the bulb. The Voss operation is an example of this type of approach. In the second method, the bulb is approached along the base of the skull from the outside. The Grunert and the Piffl operations are examples of this type. There are numerous modifications of these methods.

The best drainage is obtained with the Grunert and the modifications of the Grunert operation, as the sinus, bulb and jugular vein are converted into an open gutter in this operation. But it is technically the most difficult of the bulb operations.

The Voss operation has a much simpler technique. But the drainage is not quite as good, as at the end of this operation, the opened bulb lies at the bottom of a deep narrow funnel.

In the Piffl operation, in which the bulb is exposed by removal of the floor of the external auditory canal, there is danger of injury to the facial nerve. This operation can only be done after a radical mastoidectomy. The Grunert and the Voss operations can be done after either a simple or a radical mastoidectomy.

In all operations on the jugular bulb, the jugular vein must be tied off first. This is done for two reasons:

1. To prevent pieces of thrombus from being carried into the circulation during the manipulations on the bulb.

2. To drain the bulb from below.

Grunert Operation. Grunert's description of his operation on the bulb follows:

"After operation on the sinus and jugular ligation, the mastoid and neck incisions are joined. The soft tissues are separated from the base of the skull up to the jugular foramen [Fig. 98]. The transverse process of the atlas sometimes narrows the operative field and may have to be resected [Fig. 99]. In 12 cases which were operated on, the transverse process of the atlas was resected four times. The last bridge of bone between the lower end of the sinus and the jugular foramen is removed with a chisel or Luer bone forceps, after the rectus capitis lateralis is removed from its attachment to the jugular process of the occiput.

"If a chisel is used, it is important to place the chisel parallel to the median plane. The bony bridge is divided at its medial end. The next chisel cut is lateral and parallel to the first, at a distance of 1 cm. from the latter. This avoids injury to the facial nerve at its exit from the stylomastoid foramen. The distance between the medial chisel cut and the stylomastoid foramen is always more than 1 cm. When 1 cm. of the outer wall of the jugular foramen is removed, the bulb is in full view. If this does not give sufficient exposure, some more bone may be removed, the direction of the chisel strokes being oblique from behind and above, forward and downward; that is, divergent from the facial canal. The bulb is then incised, and its outer wall excised [Fig. 100]. After the thrombus is removed, the openings of the vessels which empty into the bulb are inspected, especially the inferior petrosal sinus. If

FIG. 98. GRUNERT OPERATION ON JUGULAR BULB. MASTOID INCISION AND NECK INCISION CONNECTED. SIGMOID SINUS UNCOVERED AS LOW DOWN AS POSSIBLE, AND OPENED.

C, carotid artery; D, posterior belly of digastric muscle; F, facial vein; IJ, internal jugular vein; P, parotid gland; S, sigmoid sinus; SA, spinal accessory nerve; SM, sternomastoid muscle.

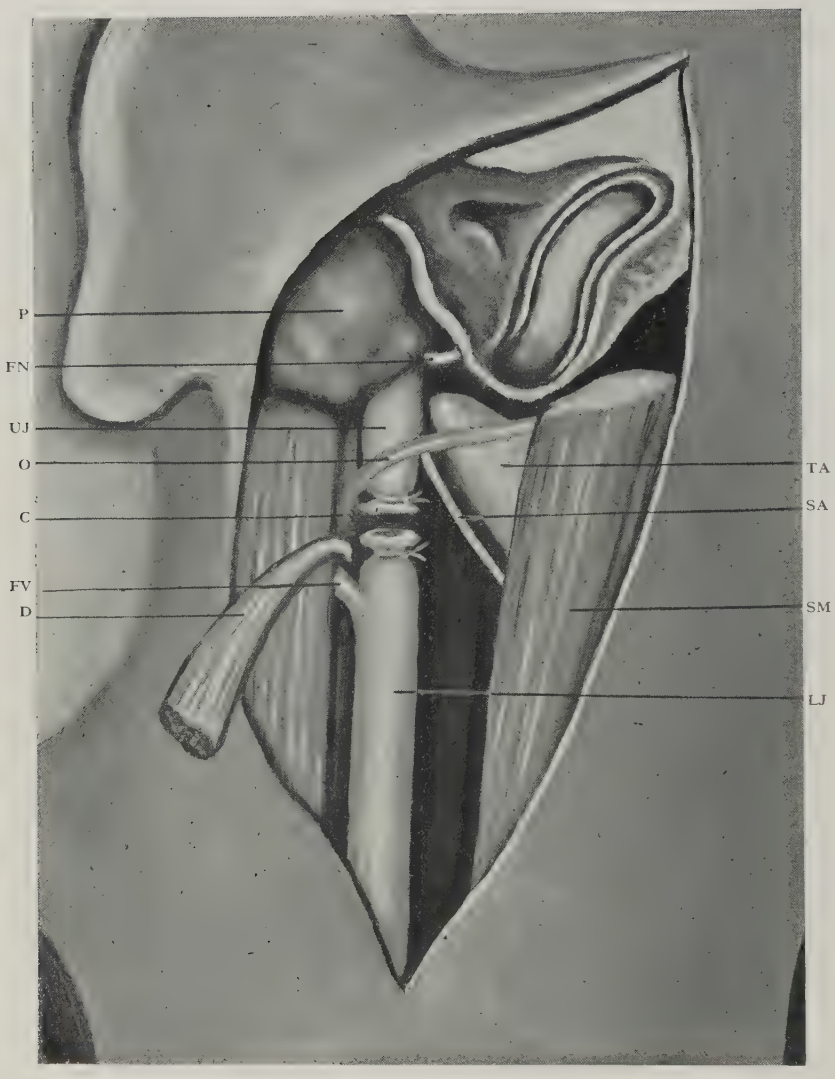

FIG. 99. GRUNERT OPERATION ON JUGULAR BULB. STERNOMASTOID MUSCLE SEPARATED FROM MASTOID TIP. DIGASTRIC MUSCLE SEPARATED FROM DIGASTRIC GROOVE AND THROWN FORWARD. INTERNAL JUGULAR VEIN DOUBLE-LIGATED AND DIVIDED BETWEEN LIGATURES.

C, carotid artery; D, posterior belly of digastric muscle; FN, facial nerve; FV, facial vein; LJ, lower end of the internal jugular vein; O, occipital artery; P, parotid gland; SA, spinal accessory nerve; SM, sternomastoid muscle; TA, transverse process of atlas; UJ, upper end of internal jugular vein.

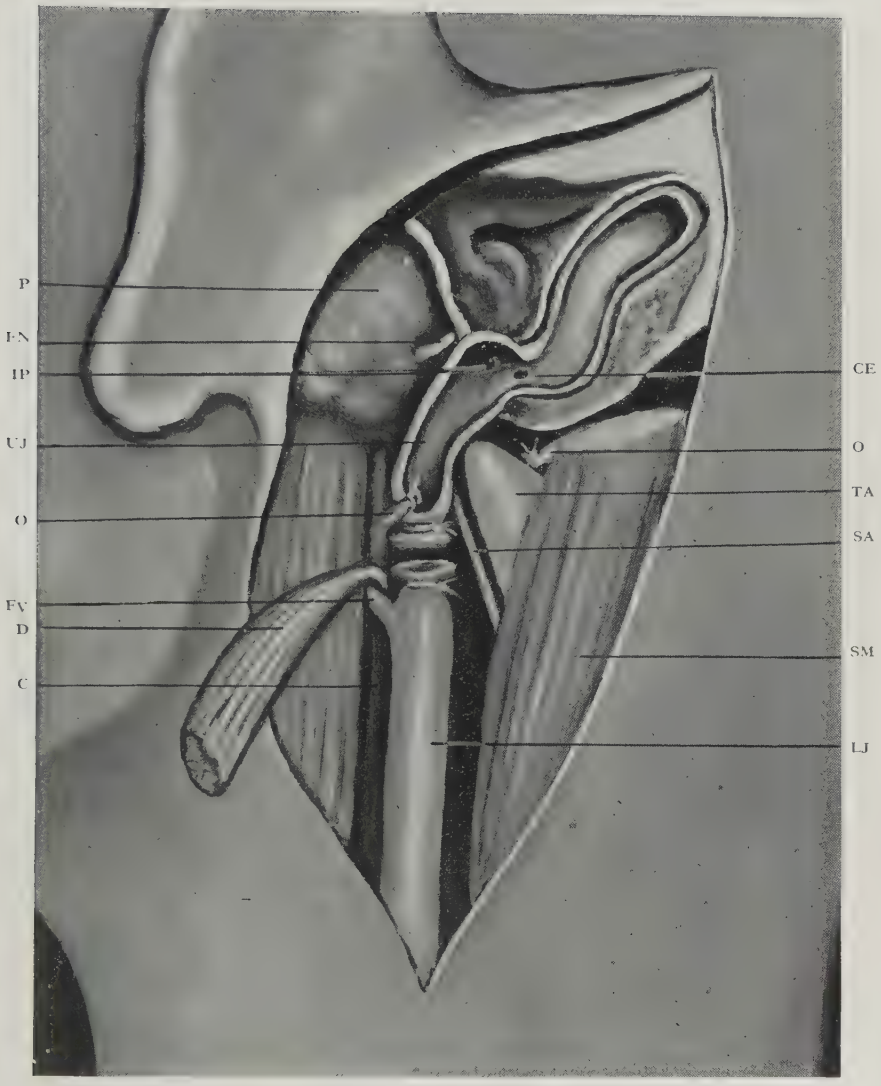

FIG. 100. GRUNERT OPERATION ON JUGULAR BULB. OUTER RIM OF JUGULAR FORAMEN REMOVED. JUGULAR BULB AND UPPER PART OF INTERNAL JUGULAR VEIN SPLIT OPEN, CONVERTING SINUS, BULB AND VEIN INTO AN OPEN GUTTER.

C, carotid artery; CE, opening of condyloid emissary into jugular bulb; D, digastric muscle; FN, facial nerve; FV, facial vein; IP, opening of inferior petrosal sinus into bulb; LJ, lower end of internal jugular vein; O, occipital artery ligated; P, parotid gland; SA, spinal accessory nerve; SM, sternomastoid muscle; TA, transverse process of atlas; UJ, upper part of internal jugular vein opened.

there is a thrombus in its mouth, it should be removed. Examination should be made for a possible peribulbar abscess.

"If the jugular vein contains a thrombus, the vein should also be split and cleaned out. The sinus, bulb and jugular vein then constitute an open gutter.

"In order to expose the jugular vein, the sternomastoid and digastric muscles are separated from their attachment to the mastoid, and pushed to one side, and the stylohyoid muscle is divided. The spinal-accessory and hypoglossal nerves must not be injured. The spinal accessory is behind the jugular vein, and the hypoglossal is in front of the vein.

"The occipital artery and the posterior auricular artery, which are branches of the external carotid, pass backward and upward, crossing the jugular vein obliquely. Both arteries must be double-ligated and divided.

"If the perivascular tissue is infiltrated or edematous, the operation is more difficult. A sound may be inserted into the vein above the point of ligation and passed up through the vein, thus acting as a guide in the isolation of the vein."

The dangers of the Grunert operation are injury to the facial nerve and to the vertebral artery. Injury to the vertebral artery may occur when the transverse process of the atlas is resected. If the jugular foramen is opened from the outer side, there is no danger of injury to the ninth, tenth and eleventh nerves, as these nerves lie in the internal and anterior part of the jugular foramen.

Voss Operation. The technique of the Voss operation on the jugular bulb is described by him:

"After performing the mastoid operation, the sigmoid sinus is uncovered in the usual way. The sinus is followed down as far as possible. As the lowest part of the sinus is reached, the direction of the sinus changes from vertical to horizontal. The lateral wall of the sinus becomes its lower wall. The sinus

is opened and the clots are removed [Fig. 101]. A probe is passed into the bulb. The firm anterior wall of the jugular fossa can be felt with the probe. The length of the probe which is necessary to reach the anterior wall of the

FIG. 101. VOSS OPERATION ON JUGULAR BULB. SINUS UNCOVERED DOWN TO POINT WHERE OUTER SINUS WALL BECOMES LOWER WALL.

fossa determines whether the lowermost part of the sinus has actually been reached. With a bent probe, the other walls of the jugular fossa may be felt. The posterior wall of the bulb is next removed. The bony wall above the lowermost part of the sinus is removed with a chisel, layer by layer, for a width of ½ cm. This work is done medial to the descending portion of the facial nerve. Care must be taken not to go too high, in order to avoid injury to the posterior semicircular canal [Figs. 102 and 103]. In this way, the bulb

is opened from behind. The opening into the bulb can be enlarged at will. The membranous wall of the bulb is then removed."

The principal disadvantage of this operation, as compared to Grunert's,

FIG. 102. VOSS OPERATION ON JUGULAR BULB. REMOVAL OF POSTERIOR WALL OF BULB, BELOW POSTERIOR SEMICIRCULAR CANAL AND MEDIAL TO FACIAL NERVE.

is that the jugular vein is not included in the operation. The operation is much simpler than Grunert's, and there is less danger to the facial nerve.

Piffl Operation. The Pifffl operation consists in the removal of the lower and anterior wall of the external auditory canal, and the floor of the middle ear, thus exposing the bulb and the upper part of the internal jugular vein.

The technique of the operation is as follows:

"After a radical mastoid operation, the mastoid incision is extended downward a few centimeters, and the periosteum is removed from the lower and anterior surfaces of the os tympanicum to the Glaserian fissure [Fig.

FIG. 103. VOSS OPERATION ON JUGULAR BULB. VIEW OF ANTERIOR MASTOID LAMINA FROM BEHIND FORWARD.

A, aditus; F, facial nerve; P, posterior semicircular canal; X, portion of bone below posterior semicircular canal and medial to facial nerve, which must be removed in order to expose jugular bulb from behind. The right hand figure shows this piece of bone removed.

104]. The lining of the lower and anterior bony canal walls is removed. The soft tissues are retracted forward. With a Luer bone forceps, the lower and anterior walls of the external auditory canal are removed as far as the hypotympanum. If this does not give sufficiently free access, the styloid process is separated from the muscles which are attached to it, and removed. This usually brings the upper part of the internal jugular vein into view. The bulb is now exposed by removing the outer wall and part of the roof of the jugular fossa, and the inner wall of the middle ear, with a bayonet-shaped bone forceps [Fig. 105]. It is dangerous to go too far in front of the external auditory canal, as there is danger of injuring the internal carotid artery."

The relation between the jugular fossa and the external auditory canal is not constant. Sometimes the jugular fossa is behind the plane of the external auditory canal, and sometimes it is anterior to the plane of the

FIG. 104. PIFFL OPERATION ON JUGULAR BULB. RADICAL MASTOID OPERATION DONE. SIGMOID SINUS UNCOVERED AND OPENED. PERIOSTEUM REMOVED FROM LOWER AND ANTERIOR SURFACES OF OS TYMPANICUM TO GLASERIAN FISSURE.

canal. The conditions are most favorable for doing a Piffl operation, when the jugular fossa is opposite the external auditory canal. When the jugular fossa is anterior to the canal, there is danger of injuring the facial nerve. When the jugular fossa is posterior to the canal, there is danger of injuring the carotid artery.

TANDLER MODIFICATION OF GRUNERT OPERATION. Tandler modified the Grunert operation. In his operation, he keeps the facial and the spinal

accessory nerves in view during the entire operation. In this way there is less likelihood of injuring these nerves. The Tandler operation is performed as follows:

FIG. 105. PIFFL OPERATION ON JUGULAR BULB. LOWER AND ANTERIOR WALLS OF EXTERNAL AUDITORY CANAL REMOVED. OUTER WALL OF JUGULAR BULB REMOVED.

C, internal carotid artery; J, jugular bulb opened; S, styloid process.

The retroauricular and the jugular skin incisions are joined. The upper part of the sternomastoid muscle is exposed, and turned back. The spinal accessory nerve is seen on the inner surface of the muscle, passing from above and in front, downward and backward. A thread is loosely tied to the nerve, so that it can be kept in full view during the operation. The anterior lip of the wound is retracted forward with the parotid gland, after separating the gland from the sternomastoid muscle. If the finger is passed along the anterior

border of the sternomastoid muscle up to the base of the skull, the tip of the finger reaches the space between the tip of the mastoid process and the styloid process. The stylomastoid foramen with the exit of the facial nerve is exposed here, and kept in view during the entire operation. The tip of the mastoid process is removed, and the attachment of the sternomastoid to the mastoid tip is pulled back. This brings into view the posterior belly of the digastric muscle. This muscle is separated from the digastric fossa and turned downward and forward. The exit of the facial nerve from the stylomastoid foramen is now plainly seen. The occipital artery is double-ligated and divided. The jugular vein is separated from the surrounding tissues, from the point of ligation above the entrance of the facial vein up to the jugular fossa. The edge of the jugular foramen can be felt with the finger. The rectus capitis lateralis muscle covers the upper part of the vein. It is separated with the periosteum from the base of the skull. This exposes the lateral margin of the jugular foramen. If the sigmoid sinus has been uncovered as low down as possible, there remains only the narrow bony margin of the jugular foramen, which can be removed very easily with a chisel. This exposes the wall of the bulb. The entire vein, bulb and sinus are split, completing the gutter operation. Tandler does not find it necessary to resect the transverse process of the atlas.

Whiting Operation. Whiting has recently described an operation for reaching the bulb through the mastoid wound.

"After the sigmoid sinus has been uncovered as far down as possible, a vertical line is made on the convexity of the sigmoid groove just below the knee of the sinus, and is extended thence downward through the occipital groove, and into the jugular process of the occipital bone; all the included bone is removed and a moderately wide surface of the cerebellum exposed posteriorly. This gives ample space in which to cut away the bone forward toward the jugular bulb, either with the chisel or the bent rongeur. This

measure permits approach to the bulb. The next step is to remove with a curette all the diploic structure which lies immediately below the posterior semicircular canal. You then have the jugular bulb immediately beneath a thin layer of bone. Then with a chisel break through that layer of bone, and you are in the jugular bulb. The lower limb of the posterior semicircular canal is slightly below the level of the floor of the tympanum. Consequently when you have fully exposed this structure, you are immediately over the bulb, and you can easily go into it. In enlarging the opening of the bulb, care must be exercised not to injure the aqueductus vestibuli."

Neumann advises the use of a Gigli saw for operation on the bulb. The saw, covered by a Nélaton catheter, is passed into the open sinus, and out through the peripheral end of the jugular vein. The catheter is then removed, and the bone is sawed through from within outward.

If a thrombus is present in the inferior petrosal sinus, only its posterior end can be reached by operation. A Grunert or Tandler operation is done on the jugular bulb. This brings into view the opening of the inferior petrosal sinus into the bulb. If there is a thrombus at this point, it can be removed with forceps. Suction may be effective if the thrombus is broken down. In cases where a thrombus in the inferior petrosal sinus extends into the cavernous sinus, the cavernous sinus and the bulb should be opened, and the inferior petrosal sinus washed through from the bulb to the cavernous sinus.

The superior petrosal sinus can be reached by removal of the tegmen antri and tegmen tympani. The posterior half of the superior petrosal sinus can be uncovered in this way. In order to reach the anterior portion of the sinus, part of the petrous pyramid must be removed.

If there is a thrombus in a persistent petrosquamous sinus, it is reached by a removal of the roof of the antrum and middle ear.

A thrombus in the superior longitudinal sinus is reached by a removal of the cortex of the skull over this sinus. Hirsch and Schwab reported a case of

thrombosis of the superior longitudinal sinus, secondary to thrombosis of the lateral sinus, which they cured by operation.

No case of thrombosis of the inferior longitudinal sinus or of the straight sinus has ever been attacked operatively.

Operations on Cavernous Sinus

The cavernous sinus can be reached by several routes. But every method of approach to this sinus is fraught with great difficulty and considerable danger. On account of the numerous septa dividing the lumen of the cavernous sinus into small chambers, it is almost impossible completely to evacuate the contents of the sinus by operation, and to obtain adequate drainage. The fact that thrombosis of the cavernous sinus does not long remain unilateral, makes operative interference of very little value, unless it is done very early in the disease. In all the extensive literature of this disease, only 2 cases of cure by operation have been reported, one by Bircher and one by Eagleton. A number of cases, however, are cured without operation. Dwight and Germain found that 7 per cent of cases of cavernous sinus thrombosis recover spontaneously. The writer observed a case of cure without any operative interference. Adair Dighton reported a cure with operation on the sigmoid sinus, jugular bulb and internal jugular vein, without operation on the cavernous sinus.

Eagleton divides the cases of cavernous sinus thrombosis into the acute fulminating cases and the chronic, slowly obliterating cases. Only in the latter type is there any hope of recovery.

There are three methods of approach to the cavernous sinus:

1. Through a temporal flap.
2. Through the orbit.
3. Through the nose and sphenoidal sinus.

The method of approach through a temporal flap is indicated in cases where the cavernous sinus thrombosis is due to an extension from the ear and petrosal sinuses.

The method of approach through the orbit is indicated in cases where the extension to the cavernous sinus is from the face through the orbit, and where the eye is the seat of extensive involvement.

The method of approach through the nose and sphenoidal sinus is indicated in cases where the extension to the cavernous sinus is through the sphenoidal sinus.

The technique of the method through a temporal flap is very much like that for operation on the Gasserian ganglion.

A semicircular incision is made through the skin and soft tissues down to the bone. It starts at the level of the zygoma, just above the anterior margin of the auricle, extends upward, and then forward and downward, ending at the same level somewhat external to the outer margin of the orbit. The base of the flap is 4 cm. long. Its greatest width is 5½ cm., and its height is 6 cm. The periosteum is pushed back for a short distance, and the incision carried through the bone. Along the line of incision, four trephine openings are made and the bone divided with a Gigli saw between these openings, after the dura is separated from the overlying bone, by means of a narrow flexible flat elevator (Fig. 106). The bone-flap, attached to the overlying soft tissues and skin, is broken through at its base. A ridge of bone about 1 cm. high is removed below the line of fracture, in order to obtain better access to the floor of the middle fossa of the skull.

The dura is separated from the floor of the middle fossa by blunt dissection. The middle meningeal artery is ligated. The temporosphenoidal lobe of the brain covered by dura is lifted up with a broad blunt retractor, until the cavernous sinus is brought into view, lying medial to the Gasserian ganglion and the first and second branches of the latter (Fig. 107). The sinus

is incised, the contents evacuated and a drain inserted into the sinus, and carried out through the lower angle of the wound. The bone and skin flap is replaced and the skin sutured.

FIG. 106. OPERATION ON CAVERNOUS SINUS. INCISION FOR TEMPORAL FLAP THROUGH SKIN, MUSCLE AND PERIOSTEUM. FOUR TREPHINE OPENINGS THROUGH BONE, AND BONE DIVIDED BY GIGLI SAW BETWEEN TREPHINE OPENINGS.

Hartley operated on a case of aseptic thrombosis of the cavernous sinus by this method. The patient lived for two and a half months, and died of an extension of an orbital sarcoma.

Voss advises removal of the bony portion of the temporal flap. He makes a semicircular incision, of which the anterior portion begins at the angle of the zygoma, and the posterior portion ends a finger's breadth behind the

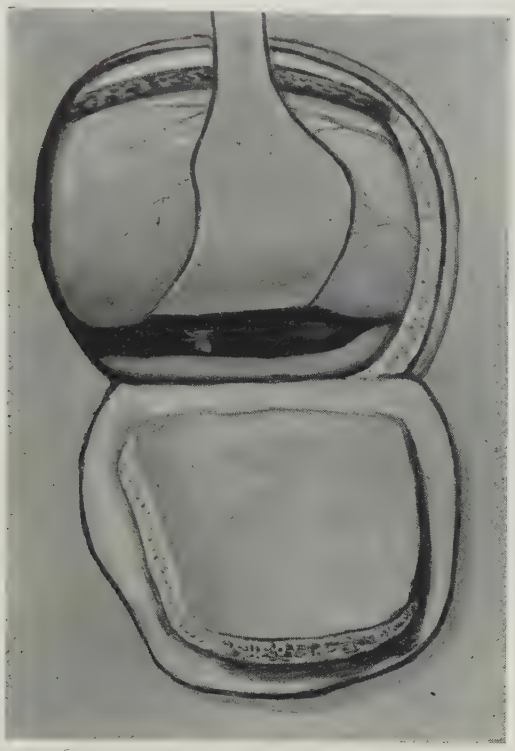

FIG. 107. OPERATION ON CAVERNOUS SINUS. BONE-MUSCLE-SKIN FLAP TURNED DOWN. BRAIN RETRACTED UPWARD AND MIDDLE MENINGEAL ARTERY TIED.

ear. The zygoma is divided behind with a saw and broken through with a chisel in front. Then the skin-muscle-periosteal flap is separated from the skull from above downward. The bone is removed with chisel and rongeur down to the foramen ovale, so that the third division of the fifth nerve is exposed. The bone is then removed a little further forward, close to the foramen rotundum. This brings the second branch of the fifth nerve into view. The patient is then placed in a sitting posture in order to stop the

venous hemorrhage. A lumbar puncture is done to allow the brain to fall back. The brain is retracted with a broad retractor, upward, until the anterior part of the Gasserian ganglion is exposed. The cavernous sinus lies

FIG. 108. OPERATION ON CAVERNOUS SINUS THROUGH TEMPORAL FLAP AND ORBIT. TEMPORAL FLAP OPERATION COMBINED WITH KROENLEIN OPERATION, EXPOSING ORBITAL CONTENTS, AND ALLOWING ORBITAL VEINS TO BE FOLLOWED INTO CAVERNOUS SINUS.

OR, outer rim of orbit.

medial to the ganglion and its first and second branches. In order to open the sinus, the knife must be directed basally, and the dura above the sinus

must not be wounded. A tampon or cigarette drain is introduced and the wound closed up to its posterior angle. If the middle meningeal artery is large, it should be ligated as soon as it comes into view.

If the eye and not the ear is the origin of the cavernous sinus thrombosis, a Kroenlein operation can be combined with the above described operation. The posterior part of the incision remains the same. The anterior part, instead of being vertical, curves forward and upward, so that it passes in front of the outer margin of the orbit to the edge of the lids. The zygoma is divided through the lower end of the incision. The orbital periosteum is removed from the outer wall of the orbit, and a portion of this wall is resected with chisel or saw according to Kroenlein. The fragment of bone remains attached to the muscle flap. The remaining portion of the outer orbital wall is removed with bone forceps up to the optic foramen. Then the cranial cavity is opened as before. The portion of bone which remains between the two openings, that is, the outer margin of the optic foramen, is removed with a chisel. The orbital periosteum is split horizontally, the plexus of veins which leads from the orbit to the cavernous sinus is followed until the latter is reached (Fig. 108). The simultaneous opening of the orbit allows drainage of the latter, as there are frequently numerous abscesses in the orbital connective tissue.

Langworthy has described an operation on the cavernous sinus through the sphenoidal sinus, which he performed on the cadaver a number of times. It is done as follows:

"The posterior naris is plugged on one side. The ethmoidal labyrinth middle turbinate and anterior sphenoidal wall are removed with the curette, forceps, punch and long gouge and hammer. After the sphenoid is opened, a curette is pushed through its roof, close to the junction of the roof and external wall. The blunt end of the curette will push the carotid artery aside. By rotating the curette in a forward direction, away from the carotid artery,

a hole of some size can be made leading directly into the cavernous sinus. This can be enlarged by a long narrow chisel. The danger in this operation is not in the thick-walled large carotid artery, but rather in some small

FIG. 109. OPERATION ON CAVERNOUS SINUS THROUGH SPHENOID SINUS. ANTERIOR AND LOWER WALLS OF SPHENOID SINUSES REMOVED AFTER DOING SUBMUCOUS SEPTAL RESECTION. SEPTUM BETWEEN TWO SPHENOID SINUSES REMOVED. OPENING MADE THROUGH OUTER PART OF ROOF OF SPHENOID SINUS, GIVING ACCESS TO CAVERNOUS SINUS.

branches which are frequently given off in this region, which may be accidentally torn. Another danger is in opening a partially thrombosed sinus."

The sphenoidal sinuses may be opened after a submucous septal resection of the nose as described by Hirsch for the removal of a hypophyseal tumor. He removes the entire septum, leaving the mucous membrane covering on both sides. Then working between the two flaps, thus avoiding the likelihood of infection from the nasal cavities, he strips the mucous membrane from the anterior surface of the two sphenoids and removes the bony anterior wall, the septum and the roof of the sphenoids.

Cushing advises drawing up the upper lip, and making a transverse incision across the labial frenum, about 2 cm. in length. This is carried down to the nasal spine of the superior maxilla. With blunt dissection, the soft parts are pushed back on each side from the bony nasal opening, until the cartilaginous septum is exposed. The mucous membrane is separated from

the septum, and the entire cartilaginous and bony septum is removed. The two flaps of mucous membrane are retracted by deep smooth retractors, or a bivalve speculum. The anterior and lower walls of the sphenoids are removed,

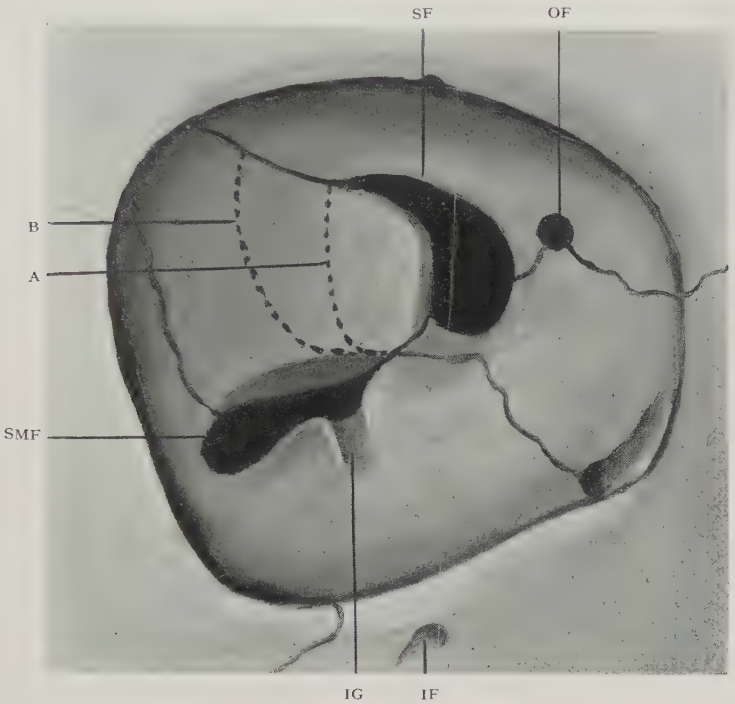

FIG. 110. OPERATION ON CAVERNOUS SINUS THROUGH ORBIT. ORBITAL CONTENTS REMOVED. PERIOSTEUM REMOVED FROM POSTERIOR HALF OF FLOOR OF ORBIT. (*After Mosher.*)

A, first bone incision; B, bone window enlarged outward to this point; IF, infraorbital foramen; IG, infraorbital groove; OF, optic foramen; SF, sphenoidal fissure; SMF, sphenomaxillary fissure.

and the septum between the two sinuses removed. The roof of the sphenoidal cells is then removed, and the cavernous sinus opened (Fig. 109).

In cases of cavernous sinus thrombosis, in which there is marked involvement of the eye, Mosher advises attacking the cavernous sinus through the orbit. His description of the operation follows:

"The globe of the eye is removed, and the orbit cleaned out. The ophthalmic artery is tied. The periosteum is cleared from the posterior half

of the floor of the orbit, and the groove is recognized, in which the superior maxillary nerve runs. The next step is to separate the periosteum from the orbital surface of the great wing of the sphenoid, and to recognize the outer end of the sphenoidal fissure. With a chisel placed vertically, a cut is made through the vertical plate of the great wing of the sphenoid from the notch for the superior maxillary nerve below, to the outer end of the sphenoidal fissure above [Fig. 110]. The bone is thin along this line and is readily removed. It is important to make sure that the whole of the bone making the lower border of the fissure is taken away. The bone opening is enlarged outward ½ cm., using either rongeur or chisel. The lower edge of the bone window should be brought flush with the floor of the orbit. Unless this is done, it is not easy to get at the bottom of the middle fossa from which the dura is elevated. If the window is carried further outward the zygomatic fossa is opened.

"The bone window made and enlarged outward and brought flush with the floor of the orbit, the dura is then elevated from the floor of the middle fossa, working from the outer boundary of the bone window inward. On the cadaver the dura can be separated from the outer wall of the cavernous sinus for a distance backward of about 1 cm. Then the separation becomes hard. If the elevation is persisted in at the level of the bottom of the bone-window, a pin-head opening is torn in the outer wall of the sinus at this point. Above and beyond this point of adhesion between the outer wall of the cavernous sinus and the dura, the two can be separated for about 1 cm. further. Then the emergence of the ophthalmic division of the fifth nerve from the Gasserian ganglion halts the separation. If an attempt is made to separate the inner wall of the cavernous sinus from the outer wall of the body of the sphenoid bone, the knife may have to be used to start the separation, but once it is started, it can be carried back easily to the limit of the sinus.

"Exposure of the outer wall of the sinus, though limited in extent, is the better. Choosing then the exposure of the outer wall of the sinus and having separated the dura from it for 1 cm., a blunt pointed knife is placed against

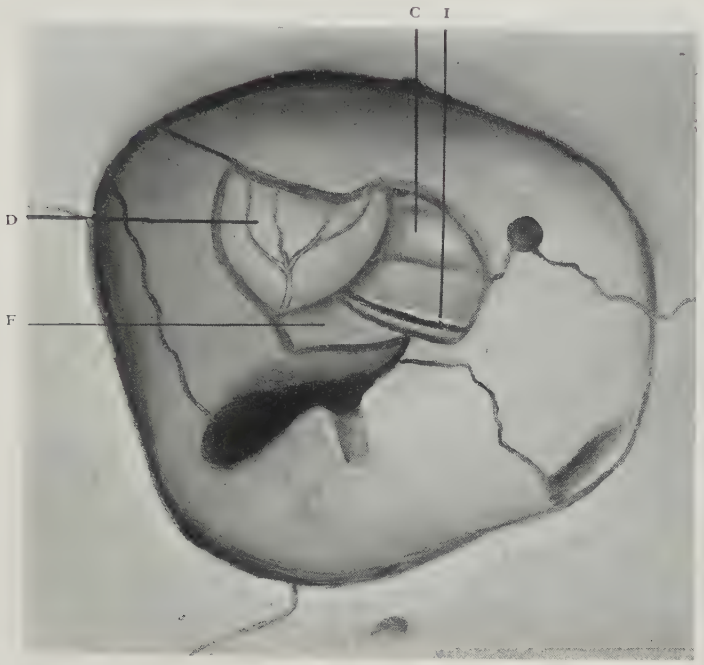

FIG. 111. OPERATION ON CAVERNOUS SINUS THROUGH ORBIT. BONE WINDOW IN OUTER WALL OF ORBIT REMOVED. CAVERNOUS SINUS INCISED. (*After Mosher.*)

C, internal carotid artery; D, brain covered by dura; F, floor of middle fossa; I, line of incision in cavernous sinus.

the outer wall of the sinus on a level with the floor of the orbit, and the knife blade carried forward toward the body of the sphenoid until it is stopped by the bone. This incision opens the wall of the cavernous sinus for 1 cm., and is well below the internal carotid artery [Fig. 111]. Through this incision into the cavernous sinus, a small curette can be carried back in the sinus to the opening of the superior and inferior petrosal sinuses, that is, through the whole body of the sinus. The distance from the rim of the orbit is on the average between 3 and 4½ inches."

Eagleton states that there are three indications in the treatment of cavernous sinus thrombosis:

1. Eradication of the original focus of infection.

2. As free drainage as possible through the path of original infection.

3. To place the inflamed and thrombosed sinus at rest by ligation of the internal or common carotid artery of the affected side.

The pulsation of the carotid artery in the cavernous sinus causes a certain amount of movement in the inflamed tissues and interferes with healing. Preliminary ligation of the carotid artery not only furnishes rest to the parts, but renders operative hemorrhage negligible. The tying of the internal carotid is just as effective as tying the common carotid in furnishing rest to the inflamed tissues, but it does not control surface hemorrhage, and takes longer to perform than tying the common carotid.

Bircher cured a case of cavernous sinus thrombosis by removal of the petrous pyramid. He made two trephine openings above the external auditory canal, and opened the middle fossa through these openings. The petrous pyramid was then removed with a chisel and bone forceps. As the apex of the pyramid was removed, an abscess cavity was opened. The opening was enlarged with a curette, and necrotic tissue was removed with scissors. The operation was followed by a facial paralysis. This operation would be indicated only if there is necrosis of the petrous pyramid.

Loss of Blood

As there is frequently considerable loss of blood in operations on the venous sinuses of the skull, the patient may go into collapse during the operation, or immediately after. An apparatus for intravenous saline infusion should always be ready in the operating room, and if the pulse becomes very rapid and feeble, an intravenous infusion of normal saline should be given. In 2 cases in which collapse occurred on the table, and in 1 case in which it

occurred about fifteen minutes after the patient left the operating room, this procedure was resorted to by the writer. So alarming was the condition of these patients, that the writer feels confident that they would have died, had the infusion not been promptly given. They all reacted promptly to this treatment. If there is any difficulty in entering a vein, hypodermoclysis may be done instead of intravenous infusion.

Blood Transfusion

Blood transfusion is a very valuable adjuvant in the treatment of otogenic general sepsis. The transfusion may be done at the time of the sinus operation, or several days later. It may be repeated several times, 200 to 500 c.c. of blood being used, according to the age and size of the patient. The transfusion not only restores the amount of blood in the circulatory system, but it also combats the anemia which is always present in these cases, and increases the bactericidal property of the blood.

In some cases of otogenic general sepsis, in which the symptoms are not very severe, the condition may be cured by blood transfusion without operation on the sinus.

Intravenous Use of Mercurochrome

The intravenous use of mercurochrome 220 soluble (dibromoxymercuri-fluorescein) in a few cases of otogenic general sepsis has met with apparently favorable results. Young, Hill and Colston treated several cases of septicemia successfully in this way. A 1 per cent solution of the drug is used. From 1 to 5 mg. of the drug per kilogram of body weight should be used. This may be repeated once or twice or even three times at intervals of one or two days.

When large numbers of bacteria are free in the circulation, smaller doses of the drug should be used, as the reactions following the use of the

drug are not caused by the mercurochrome but by the liberation of toxins from the killed bacteria in the circulation. If the dose is repeated, it should be increased or decreased according to the initial reaction. Tobey advises giving a 1 mg. per kilogram dose.

Hedges reported 2 cases of cure in sinus thrombosis, one by operation and mercurochrome, and one by mercurochrome alone. The first case had 175 mg. of mercurochrome and the second case had 250 mg. Porter reported 2 cases of sinus thrombosis and 1 case of sepsis cured by mercurochrome in conjunction with surgical treatment.

In order to get results with mercurochrome, the patient must have some resistance. The greater the reaction caused by the injection, the more favorable is the result.

There is danger in the use of mercurochrome when there is kidney involvement. The use of this drug is still in the experimental stage, and it should be used with great care, as we do not yet know all the possible dangers connected with its use.

Treatment of Metastases

Metastases must be carefully watched for, and promptly treated. Areas of infiltration in the skin and subcutaneous tissues frequently resolve under wet dressings. When fluctuation occurs, the abscess must be incised, and drained. Redness and swelling of the joints frequently subsides with immobilization of the joint and wet dressings. When an abscess forms in the periarticular tissues, it should be incised and drained. When fluid occurs within the joint, it should be aspirated. Some cases are cured by aspiration and irrigation. If this does not help, the joint may have to be opened. Abscesses in the lung or pleural cavity should be treated by proper surgical means. Abscesses in the liver, spleen, kidneys or intestines may have to be treated surgically.

Conclusion

There are no contraindications to the operation for sinus thrombosis. Patients with numerous and extensive metastases, and even with cerebral symptoms sometimes get well. Operation should be undertaken even in the most desperately ill cases. The surgeon will be rewarded by seeing some of these patients recover. But the treatment of these cases requires more patience and painstaking effort than any other class of cases that the otologist is called upon to treat.

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